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THE AMERICAN OPHTHALMOLOGICAL SOCIETY

A RETROSPECT OF SEVENTY-FIVE YEARS

HARRY FRIEDENWALD, M.D.

BALTIMORE

The American Ophthalmological Society is now completing seventy-five years of activity. The officers decided to commemorate this occasion and invited me to review this long and honorable history of scientific and professional association in the service and devotion to ophthalmology.

Scientific societies, it must be remembered, are distinctly modern institutions. They came into being with the Renaissance in the seventeenth century in Rome and Florence. Similar societies soon appeared in London and in Oxford; those in Oxford combined and formed the Royal Society of London, which received its charter in 1662; the French Academy of Sciences came into being in 1666. The oldest Medical Society of London was founded in 1773, the Royal Medical and Chirurgical Society in 1805 and the British Medical Association in 1832.

The American Medical Association was called into being in 1847.

These facts have been told in order to bring out the relatively early period of the founding of the American Ophthalmological Society in 1864. It was the first society devoted to a special branch of medicine in this country, and it was founded sixteen years before the Ophthalmological Society of the United Kingdom (in 1880).

But it is necessary to consider not only the time at which, but also the conditions under which, the American Ophthalmological Society was founded. The state of medicine was indicated in 1847 when the American Medical Association was founded to "improve the disgraceful status of medical education in the United States." An interesting description of the state of ophthalmic practice in the middle of the century was made by one of the most eminent American surgeons, Dr. Samuel D. Gross (1805-1884), who was a delegate to the first International Ophthalmological Conference, held in Brussels in 1857. He had been invited

This address was given by request at a banquet at Hot Springs, Va., held on the evening of June 6, 1939, to celebrate the seventy-fifth anniversary of the American Ophthalmological Society.

to make the report on ophthalmology in America. The following quotation is a translation from his address:¹

The study of the diseases of the eye has not received in this country the attention which it deserves. In Philadelphia we have a hospital especially devoted to their treatment, and there are excellent ophthalmic infirmaries in New York, Boston and a number of other larger cities. But in the interior of the country these diseases are greatly neglected or treated exclusively by quacks.

The ophthalmoscope is used here and there by some few practitioners. My friend Dr. E. Williams, of Cincinnati, is perhaps the one who uses it most among us; it is also frequently used by Dr. A. Hewson, a distinguished physician of this city [Philadelphia] and one of the surgeons of Wills' Hospital, where they treat diseases of the eye.

The important foundation which the establishment of ophthalmic hospitals in the twenties in New York, Philadelphia, Boston and elsewhere formed for the development of ophthalmology in this country cannot be left without further consideration. There is no better proof of this foundation than that one finds the founders of these hospitals, such as John Jeffries and Edward Reynolds, of Boston, and Isaac Hays, of Philadelphia, among the small group of honorary members elected in the early years of the American Ophthalmological Society, and foremost of all, Edward Delafield, then a man of 70, who was elected as president at the first meeting of the society. One can well agree with the statement of Dr. Reynolds² that Edward Delafield and Kearney Rogers, his colleague in establishing the New York Eye and Ear Infirmary, were the "fathers of American ophthalmology"; but since this is already a dual paternity one may well add a few others; notably, Reynolds himself, John Jeffries, George Frick, Isaac Hays, George B. Wood and Squier Littell. All of this group were in general practice and chiefly in surgical practice, with the possible exception of Frick. Specialism, as shall be seen, was not established in this country until after the middle of the century, for then a new day had broken. Helmholtz had invented the ophthalmoscope in 1851, and ophthalmologic stars of the first magnitude had appeared in Europe. They attracted many of the young men in this country to Germany, Austria, France, Holland and England. These, in their turn, brought back the new science and the new practice. This it was that led to the formation of the American Ophthalmological Society in 1864.

For a number of years there was no record of the conception and the birth of the society. In 1907 Alvin A. Hubbell² prepared his most useful book, "The Development of Ophthalmology in America," and

1. Gross, S. D., in *Congrès d'ophtalmologie de Bruxelles* (1857), Paris, 1858, p. 355.

2. A. A. Hubbell (*The Development of Ophthalmology in America, 1800-1870: A Contribution to Ophthalmologic History and Biography*, Chicago, American Medical Association, 1908, p. 17) cited the interesting history of the founding of the hospitals as told by Dr. Reynolds.

gave an account of the first meeting, as did likewise Dr. Jackson³ in the excellent story which he published in *Ophthalmic Literature* in 1915.

The "Account of the Origin and First Meeting of the American Ophthalmological Society" by Henry D. Noyes, published in New York in 1875 and reprinted and mailed to the members, tells the story of the inception of this society, but it is well to recall the humorous story told by Hasket Derby and published by Hubbell:⁴

The actual founder of the society was Dr. Julius Homberger, as I used laughingly to assert, to Dr. Noyes' great indignation. About 1862, a peripatetic German adventurer of this name came to New York to engage in ophthalmic practice, and started a magazine of ophthalmology. After a few numbers this probably fell stillborn, but while it lasted it disgusted reputable ophthalmologists, and the meeting called by Dr. Noyes, after consultation with me, was really to concert measures for the establishment of a magazine that should be respectable. Eight of us came together at Dr. Noyes' office on Fourth Avenue, and after well weighing the matter, decided that a society would be a better thing to found than a magazine. So we issued a call for a meeting and held it (the first) June 7, 1864. Thus we originated. Whatever became of Homberger I have no means of knowing.⁵

It was during the last year of the Civil War that 8 men met at Dr. Noyes's office⁶ on Jan. 9, 1864; they were Herman Althof, F. J. Bumstead, Hasket Derby, John H. Hinton, W. F. Holcomb, Henry D. Noyes, D. B. St. John Roosa and Henry B. Sands, who was chosen

3. Jackson, E.: *Ophth. Lit.* 5:1 (Jan.) 1915. The Army Medical Library informed me (August 1938) that "This library does not possess the transactions of the first meeting of the American Ophthalmological Society. We have a letter dated Feb. 29, 1872, addressed to Dr. Billings and signed by H. D. Noyes, then recording secretary of the society, which states that no account of the first meeting held in 1864 was printed." The minutes of the meeting of 1874 state that Dr. Noyes was requested to "prepare a preface for the first volume, which shall include a history of the inception and original organization," etc. This was carried out by Dr. Noyes, and a copy can be found in the library of the Academy of Medicine of New York. Its great rarity is the justification for republishing it.

4. Hubbell,² p. 166.

5. There is an interesting biographic sketch of Homberger in Kelly and Burrage's "American Medical Biographies" (Baltimore, The Norman, Remington Co., 1920, p. 550), written by Shastid. Homberger was born in Germany. He spent some time in Würzburg and also in Paris, where he was assistant to Sichel. He settled in New York in 1861. He founded the American Journal of Ophthalmology in 1862, which was published bi-monthly. Volume 1 is complete, but only two numbers of volume 2 appeared. He then became "eccentric," advertised extensively and later was expelled from the American Medical Association in 1868. He became insane and entered a retreat, the "Louisiana Retreat," where he died. Lloyd has published an interesting account of Homberger's journal (*Am. J. Ophth.* 15: 1123 [Dec.] 1932).

6. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 17:35, 1919. Dr. Lucien Howe presented the society with a gavel made from one of the chairs in Dr. Noyes' office.

chairman. According to Dr. Jackson, Ezra Dyer started from Philadelphia to attend the conference but was so delayed by ice in crossing the Hudson River that he arrived after it adjourned. At this meeting it was resolved "to invite Ophthalmic Surgeons from the whole country to assemble in New York at the time of the meeting of the American Medical Association, to form themselves into an American Ophthalmic Association," and a committee was appointed to carry out this purpose.

The American Medical Association met in New York on June 7-9, 1864. Its published transactions make no mention of this meeting of ophthalmologists, which took place on June 7 at the New York Eye and Ear Infirmary, Second Avenue and Thirteenth Street. It was attended by all who had been present at the original conference and the following men in addition: Ezra Dyer, John H. Dix, B. Joy Jeffries, Francis B. Sprague, Edward Delafield, C. R. Agnew, Francis Simrock, William H. Carmalt, William Stimpson and C. A. Robertson. The meeting was called to order by Bumstead. Edward Delafield was elected chairman and H. D. Noyes secretary. A committee, composed of Bumstead, Derby and Dyer, brought in a constitution, which was discussed and adopted with the name American Ophthalmological Society; in this it was stated that "the purpose of this Society shall be the advancement of ophthalmic science and art." The society met annually, and new members were added.

One may, with propriety, look on these early members of the 1860's as the founders of the society; they include those who met in 1864 and those who were elected at that meeting and in the four subsequent years. It was this body that gave form and program and that set ethical as well as scientific standards. They began by electing 6 "honorary members" of the older generation of surgeons who had rendered distinguished services in ophthalmic hospitals and in literary production. This body of founders thus numbered 45.⁷ Of these, 14 were primarily surgeons or physicians, also interested in the diseases of the eye, and 31 were ophthalmologists. A list of these founders appears in table 1.

The ages of this group of members on their entry into the society are interesting. There were a few older men. Edward Delafield was born in 1794, John H. Dix in 1812, Abram DuBois in 1810 and J. F. Noyes in 1817. The following men were born in the twenties: H. W. Williams, 1821; E. Williams, 1822; D. D. S. Roosa, 1826; Arthur Matthewson, 1827, and Edward L. Holmes, 1828. Twenty-six were born between 1829 and 1839. (Concerning the others, the dates are unknown.) The period of membership of these members was generally

7. The name of William Stimpson does not reappear in the minutes and is therefore not included. There were 4 additional men elected, but as their names do not appear subsequently one may assume that they did not accept membership. They were Charles E. Hackley, A. Hewson, Simon Pollak and Bolling Pope.

very prolonged. The following 17 were members thirty years or more: H. W. Williams, thirty years; Edward L. Holmes, thirty-one; J. F. Noyes, thirty-two; Henry Noyes, thirty-seven; Russell Murdoch, thirty-seven; William Wallace Seely, thirty-eight (he died nine years later); H. Knapp, forty-two; Henry L. Shaw, forty-three; J. S. Prout, forty-three; O. F. Wadsworth, forty-four; D. B. S. Roosa, forty-four; John Green, forty-five; Benjamin Joy Jeffries, fifty-one; Arthur Matthewson,

TABLE 1.—*Founders of the American Ophthalmological Society*

Name	Year of Birth and Death	Period of Membership	Number of Papers Presented
Agnew, Cornelius Rea.....	1830-1888	1864-1888	13
Allin, Charles Mason.....	S* 1827-1880	1865-1878	R† 4
Althof, Herman.....	1835-1877	1864-1877	1
Bumstead, Freeman J.	S 1826-1879	1864-1876	R ..
Carmalt, William H.	S 1836-1929	1864-1928	3
Curtis, Edward M.	S 1874	1868-1874	..
Delafield, Edward.....	S 1791-1875	1864-1875	..
Delafield, Francis.....	S 1841-1915	1868-1879	R 2
Derby, Hasket.....	1835-1914	1864-1914	14
Dix, John H.	1812-1884	1864-1884	1
DuBois, Abram.....	S 1810-1891	1864-1891	..
Dyer, Ezra.....	1836-1887	1864-1887	5
Green John.....	1835-1913	1866-1913	35
Hall, A. D.	1833	1868-1897	R ..
Hay, Gustavus.....	1830-1909	1864-1907	15
Hildreth, Joseph H.	S	1864-1870	R 3
Hinton, John H.	S 1827-1905	1864-1873	R ..
Holcomb, William F.	S 1827-1904	1864-1870	R ..
Holmes, Edward L.	1828-1900	1866-1898	..
Hunt, W.	S 1825-1896	1868-1870	R ..
Jeffries, Benjamin Joy.....	1833-1916	1864-1916	24
Knapp, H.	1832-1911	1869-1911	39
Loring, Edward G.	1837-1888	1865-1888	16
Matthewson, Arthur.....	1837-1920	1868-1920	8
Morton, Thomas G.	S 1835	1865-1883	R 1
Murdoch, Russell.....	1839-1905	1868-1905	4
Newton, Homer G.	1836-1915	1868-1878	R ..
Noyes, Henry D.	1832-1900	1864-1900	47
Noyes, J. F.	1817-1896	1864-1896	2
Pomeroy, O. D.	1834-1902	1868-1895	R 5
Pray, O. A.	1869	1868-1869	..
Prout, J. S.	1833-1928	1868-1928	7
Rider, C. E.	1839-1909	1868-1909	1
Robertson, Charles A.	1829-1880	1864-1879	1
Roosa, D. B. St. John.....	1838-1908	1864-1908	9
Sands, Henry B.	S 1830-1883	1864-1870	R 1
Seely, William Wallace.....	1838-1913	1866-1904	R 7
Shaw, Henry L.	1911	1868-1911	..
Simrock, Francis.....	1864-1885	1
Sprague, Francis B.	1834-1921	1864-1921	..
Wadsworth, O. F.	1838-1911	1868-1912	28
Watts, Robert.....	S 1837	1869-1871	R ..
Williams, A. D.	1869-1873	R ..
Williams, E.	1822-1888	1865-1888	9
Williams, H. W.	1821-1895	1865-1895	8

* S indicates that the member was a surgeon or engaged in general practice.

† R indicates that the member resigned.

fifty-two; Francis B. Sprague, fifty-seven; Hasket Derby, sixty-one, and William H. Carmalt,⁸ sixty-five. Nine were members between twenty and thirty years. The average period of membership for the 31 members was thirty years!

8. In 1924 there was a special minute of celebration of the sixtieth anniversary of Carmalt.

It is not without significance that 10 of the 14 surgeons resigned, 2 after two years of membership, 3 after six years and 1 each after nine, eleven, twelve, thirteen and eighteen years; 4 others remained members until their deaths, 3 of them after six, eleven and twenty-seven years of membership and 1 of them, Dr. Carmalt, after the extraordinary period of sixty-five years of membership.

The larger number of ophthalmologists, 31 in all, lost but 2 members by resignation. A. D. Williams resigned after four years of membership and Homer G. Newton after ten years. This indicates clearly the rapidly declining interest of the surgeons in ophthalmology after the organization of the society. This is a subject to which I shall revert later.

Presentation of papers at the annual meetings must be regarded as of prime importance. One cannot estimate their value by the numbers contributed by individual members, but they indicate in some measure their activity and zeal. Comparing the relative activity of the two groups in this regard, one finds that 8 of the surgeons did not read papers; 2 read 1 paper each, 1 read 2 papers, 2 read 3 papers each and 1 read 4 papers; thus it is seen that 10 papers were read by 6 members. There were 7 ophthalmologists who did not read papers. Five read 1 paper each, 1 read 2 papers, 1 read 4, 2 read 5 each and 2 read 7 each; H. W. Williams and A. Matthewson each read 8 papers; E. Williams and St. John Roosa, 9; C. R. Agnew, 13; Hasket Derby, 14; G. Hay, 15; E. G. Loring, 16; B. J. Jeffries, 24; O. F. Wadsworth, 28; John Green, 35; H. Knapp, 39, and H. D. Noyes, 47. Thus 31 ophthalmologists contributed 300 papers, while 14 surgeons contributed 14 papers. This is another evidence of the overwhelming influence of the members of the "new order" over the "old"!

The founders were an interesting body in many ways. Delafield, whose chief interest was gynecology and obstetrics, had brought from London the idea of special hospitals for ocular diseases and had republished in 1825 the book of Travers on diseases of the eye. Bumstead's chief interest was in general diseases and diseases of the genitourinary tract, and Carmalt remained throughout and equally an ophthalmologist and a surgeon.

Of the ophthalmologists, one finds that almost all spent considerable periods in study abroad, with Bowman and Critchett in London, with Wilde in Dublin, with Desmarres and Sichel in Paris, with Donders in Holland, with von Graefe in Berlin and with Arlt and Jaeger in Vienna.

The proceedings and the papers presented in the early years bear evidence of the high purpose that led to the organization of the American Ophthalmological Society. A great impetus had been given by the

invention of the ophthalmoscope, by the new fields of clinical study that had thus been opened and by the studies of Donders. The inspiration brought from European luminaries infused this body with enthusiasm in the development in this country of ophthalmology from a branch of surgery into a special science. The need was felt to keep in close touch with the publications abroad, and during the early years the duty of reporting on the scientific progress abroad was regularly assigned to a member. These lengthy reports were a prominent feature of the early annual meetings. In the first of these (in 1865) Joy Jeffries reported on Donders' "Anomalies of Accommodation and Refraction," which had just appeared. A quotation from this report follows:

From remarks here and there cropping out in the Journals in reference to Donders' book, we fear that there are still deeper, "previously unsuspected strata of ignorance among so called intelligent and educated" surgeons and even oculists of our day. Let this not be said of members of this Society.

In the early years these problems engaged many of the members, and one finds important contributions on optics and on visual tests. Is it not surprising to read the following statement by Althof?⁹

I have no doubt that if we had the same facilities in the city of New York that are had in Europe for providing our patients with glasses, we could use them to very great advantage in many cases of asthenopia where now we can not!

This statement was made at the meeting of 1865, when the subject "Asthenopia Not Connected with Hypermetropia" was assigned for discussion. The report covers 46 pages. A large number of those present took part.

It appears that this was to become a regular order of the meetings, and committees were appointed to select subjects for discussion for the yearly meetings. Thus one finds in 1869 that the committee assigned the following subject, but there is no record that this or other assigned discussions took place:

Ophthalmic Therapeutics, Their Occasional Abuse. The Committee expressed the hope that the discussion within proper limits, may tend to lessen the indiscriminate use in medical practice of irritating collyria and patent drugs, as well as to restrain the employment of certain panaceas.

The discussion of operations for cataract was very active and reflected the work that had recently been done in Europe. As early as 1865 H. W. Williams reported on suture of the wound after extraction of cataract. The problem of photography of the fundus and its difficulties was discussed at the early meetings of the society (1865 and 1869).

9. Althof, in Discussion on Asthenopia, Tr. Am. Ophth. Soc., 1865, p. 52.

But no subject was of more immediate interest than that of specialization in medicine, and Dr. Noyes delivered an address on this subject at the second meeting, in 1865. It reflects so clearly the problem of that period that a part must be quoted:¹⁰

As decades pass and science and art attain greater completeness, the labor of the physician in fitting himself for his profession becomes more and more heavy. . . .

. . . the development of medical science and improvement of medical art are hindered by the magnitude to which the science has attained, by this inability of practical physicians to bestow on scientific research the time and labor which are indispensable to its best results.

Can anything be done to render the practice of medicine more directly in accord with the state of medical science, and to render the progress of medical science more rapid?

Two answers may be made to this question. First, medical science, it may be said, should be in the hands of those who may give themselves exclusively to its investigation, and leave the art to the practising physician. To some extent this has been done. . . .

But a second solution of the difficulty presents itself. . . . This solution is the subdivision of labor [i. e., specialization]. . . . [It] finds some difficulties in its application to medicine. . . .

Noyes referred to a class of "specialties" which have been

. . . vaunted in a pretentious and unworthy manner, by persons claiming almost marvelous abilities, and guilty of most unseemly conduct. Such are the corn doctors, the bone setters, the electricians, the pimple doctor, the hair restorers, the pill doctors, etc. These men having been styled specialists have caused a portion of the odium belonging to their evil practices to fall upon those who maintain an honorable position, while they cultivate only a limited field of practice. This sentiment will no longer continue when special departments of medicine fall into the hands of right-minded men.

Advertising in medical journals may be done as offensively as in any other prints; but when it consists simply in the announcement of name, address and specialty, it is difficult to see the offensive quality of the act.

His closing remarks were:

Already an era of good feeling has begun between specialists and general physicians. We see around us men in special practice on terms of cordiality and confidence with all their brethren. . . . So may it always be wherever the art of healing carries its beneficent work.

It was but three years later (1868) that the society adopted the rule:

No Member of this Society shall attach to his name in any public announcement the title of oculist or any similar title, or shall announce in print that he gives special or exclusive attention to special practice.

10. Noyes, H. D.: *Specialties in Medicine*, New York, J. Medole, 1865, pp. 59-74.

This injunction has been repeatedly considered and discussed.¹¹

The rapidity of progress can be sensed in one of the "Reports of Progress" of Joy Jeffries¹² for 1865-1866:

Ophthalmology has asserted its position in Science, which, we think, has been still more firmly fixed during the past two years. The recognition by the various Medical Schools and teachers that the student must have at least some rudiments of ophthalmic knowledge to become a "safe practitioner" is certainly a step in advance not to be lightly thought of or passed by. In this country our specialty still stands like most others, in such a position that, to learn what is known and the means of applying this knowledge, the student must still visit Europe.

He further wrote:¹³

Principally by means of a statistical table of Dr. Herman Knapp of Heidelberg, we are enabled to present to the Society a more or less complete report of forty-one ophthalmic hospitals and infirmaries in various parts of the world, thinking that it might perhaps prove interesting, and enable any of our members when traveling to know in what cities eye institutions were to be found, etc.

At the meeting of the following year Dr. Knapp was present. He took an active part in the proceedings and was elected a member. It is interesting to note that the first paper of Dr. Knapp was entitled "The Inaccuracy Introduced into the Determination of Visual Acuteness by Disregarding the Magnifying or Diminishing Power of Glasses."

Hasket Derby reported 2 cases of acute glaucoma following the instillation of atropine. Wharton Jones had alluded to the ill effects of this drug in acute glaucoma, but these appear to be the first cases reported in which atropine caused the immediate attack.

John Green read 3 papers at his first meeting in 1868 under the titles: "On the Use of Styles of Lead in Curing Diseases of the Lacrimal Sac," "On a New Series of Test Letters for Determining the Acuteness of Vision" and "On a Color Test for Astigmatism." It was Green who a little later introduced the use of atropine in the treatment of strabismus (1871).

Almost each meeting brought forth modifications of the ophthalmoscope.

It was at the meeting of 1869 that the ingenious striped test letters of Dr. Pray were presented.

It is tempting to mention the great number of subjects treated, in reviews and original articles, such as the papers of Loring on accommodation and on the interpretation of fundal reflexes, new methods by

11. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 13:22, 1912; 14:449, 1916.

12. Jeffries, B. J.: Tr. Am. Ophth. Soc., 1867, p. 5.

13. Jeffries, B. J.: Tr. Am. Ophth. Soc., 1868, p. 7.

Green for the detection of astigmatism, Hays and Jeffries' studies on vision, and color vision, but space does not permit.

I have referred to the rapid divorce of ophthalmology from surgery. There was, however, one branch of surgery to which many ophthalmologists have long been wedded—otology. As evidence of the association of the early members of the American Ophthalmological Society with otology one finds that of the 10 members present at the second annual meeting of the American Otological Society⁸ were members of the American Ophthalmological Society. For many years the two societies met at the same place and during the same week. On comparing the list of members of the two societies in 1938 one does not find a single member of the American Ophthalmological Society in the active list of the American Otological Society and only 3 in the senior list, which corresponds to the emeritus members of the American Ophthalmological Society.

One may regard the society as definitely established with the close of the sixties. In these few years the emergence of the specialty had led not only to the withdrawal of some general surgeons but, what is more significant, to the election of no more general surgeons as active members of the society. In providing me with some biographic data concerning Thomas G. Morton, of Philadelphia, Burton Chance informed me that "Morton, Ezra Dyer, Addinell Hewson and William Hunt were all with the staff at the Wills Hospital." Morton "served Wills until 1874. At his resignation he was made emeritus. His resignation brought the ending of general surgeons at Wills." This is additional evidence, local, of what has been shown as the close of an "old order." This new order became a rule, and in 1884 it found formal expression in the constitution, when Dr. Green, for the Committee on Membership, proposed to amend the constitution by adding: "Candidates for Membership shall have been engaged in the practice of Ophthalmic Surgery for at least five years." This was adopted.¹⁴

And here may be added another constitutional interpretation that has been of great benefit to the society in giving it distinguished members and two presidents: In 1878 it was resolved as "the sense of the Society, that residence in Canada was no disqualification to membership."¹⁵ It was the expression as well of the many ties of friendship that bind these two countries together.

"Reports of Progress" were continued only in 1870 and 1871. The former dealt with many important books and articles, among them

14. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 3:627, 1884.

15. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 2:374, 1878.

The society has continued to produce and publish an ever growing volume of scientific contributions. It has published reports, through special committees, engaged in the the study of the causes and prevention of blindness,¹⁸ ophthalmia of the newborn,¹⁹ trachoma,²⁰ wood

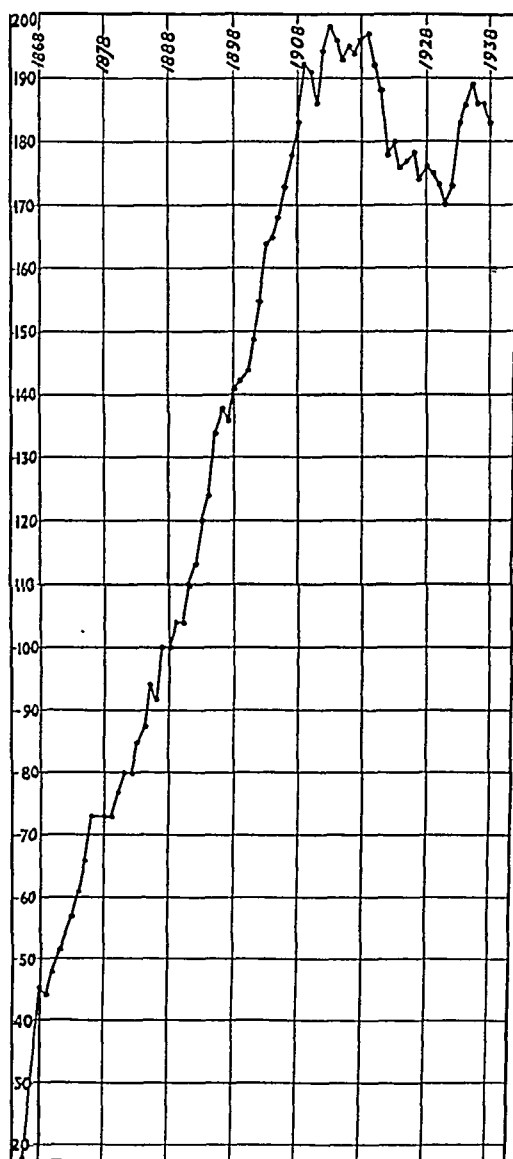


Chart showing membership of the American Ophthalmological Society from 1866 to 1938.

18. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 4:403, 1887. Report of the Committee on the Causes and Prevention of Blindness, *ibid.* 5:531, 1890. Howe, L.: *ibid.* 8:259, 1898.

19. Howe, L.: Tr. Am. Ophth. Soc. 8:52, 1897. Report of the Majority of the Committee on Resolutions Relating to Purulent Ophthalmia of Infancy, *ibid.*

alcohol blindness²¹ and compensation for injury to the eyes.²² Elaborate studies and reports have been published by its members and its committees on the standards and methods of examining the acuteness of vision, color sense, etc.,²³ and the control of color blindness and visual defects on land and sea²⁴ as well as other technical problems that required regulation.

During the Great War the society gave its fullest aid to the government. The *Transactions* of the war years contain a large number of contributions on military ophthalmology.

The society joined with other societies to aid the Census Department of the United States Government regulating the nomenclature of medical diseases.²⁵

The society has given much consideration to the problem of post-graduate instruction in ophthalmology and to the question of special degrees in ophthalmology.²⁶ In cooperation with committees of the Section on Ophthalmology of the American Medical Association and of the Academy of Ophthalmology the plan was devised "to arrange, control and supervise examinations to test preparation for ophthalmic practice." The report of the joint committee was submitted in 1915²⁷ and approved. The several members representing the three ophthalmic bodies were almost all members of the American Ophthalmological Society. Since then annual reports have been made by the board of examiners.

In 1923, in cooperation with the other national ophthalmologic bodies, a consultative Committee on Pathology was formed to aid the Army

8:265, 1898; Minority Report, *ibid.* 8:268, 1898. Minutes of the Proceedings of the Annual Meeting, *ibid.* 11:469, 1908; 12:357, 1910.

20. Minutes of the Proceedings of the Annual Meeting, *Tr. Am. Ophth. Soc.* 25:25, 1927; 27:27, 1929.

21. Minutes of the Proceedings of the Annual Meeting, *Tr. Am. Ophth. Soc.* 12:357, 1910; 12:711, 1911; 13:25, 1912.

22. Report of Committee on Compensation for Eye Injuries, *Tr. Am. Ophth. Soc.* 24:385, 1926.

23. Minutes of the Proceedings of the Annual Meeting, *Tr. Am. Ophth. Soc.* 4:17, 1885; 8:476, 1899. Report of the Committee on Standards and Methods of Examining the Acuteness of Vision, Color-Sense and Hearing, *ibid.* 9:406, 1901. Report of the Committee on Standard Test-Types, and on Reading-Tests, *ibid.* 10:189, 1903.

24. Minutes of the Proceedings of the Annual Meeting, *Tr. Am. Ophth. Soc.* 3:14, 1880; 4:17, 1885.

25. Minutes of the Proceedings of the Annual Meeting, *Tr. Am. Ophth. Soc.* 11:468, 1908.

26. Minutes of the Proceedings of the Annual Meeting, *Tr. Am. Ophth. Soc.* 13:275, 1913; 13:604, 1914.

27. Minutes of the Proceedings of the Annual Meeting, *Tr. Am. Ophth. Soc.* 14:24 and 30, 1915; 14:441, 1916.

Medical Museum in the study and diagnosis of pathologic material in the ophthalmologic field, submitted by members of the medical profession throughout the country. Dr. Verhoeff has represented the society throughout this period. It is not necessary to dwell on the scientific and educational value of this undertaking.

The relation between the American Ophthalmological Society and other national medical bodies has been most cordial and cooperative. It was on but one occasion, in the middle of the eighties, that a period of antagonism arose toward the American Medical Association because of action which the American Ophthalmological Society disapproved in preparation of the International Congress of Medicine to be held in Washington, D. C.²⁸

It has been mentioned that the American Ophthalmological Society was the first of the national societies devoted to special branches of medicine. It thus became the model for the others to follow. The first of these was the American Otological Society (1868). Others gradually appeared. The American Ophthalmological Society may properly reckon the incentive to its credit.

The American Ophthalmological Society took an active hand in the arrangements of the triannual Congress of American Physicians and Surgeons in Washington, D. C., but was unwilling to form an alliance that would interfere with its autonomy or independent meetings.²⁹ It met with its sister societies triannually from 1888 until 1933, when it withdrew.

The society is officially represented on the board of governors of the American College of Surgeons by 3 members.³⁰

The society has taken active part in arranging for international congresses.³¹

It would be of great interest to review the scientific papers that have appeared during these many years since 1870. Any selection might give the appearance of invidious distinction. However, I think that all will agree that such candor as was shown in the following incident has rarely been equaled. In 1885 Hasket Derby³² reported 2 cases of penetration of the eyeball by scissors in operations for strabismus. In the discussion Herman Knapp reported that he had pierced the sclerotic in 3 cases, and

28. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 4:18, 1885.

29. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 4:179, 1886; 4:404, 1887.

30. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 24:44, 1926.

31. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 2:123, 1874; 9:13, 1900; 10:208, 1904; 13:602, 1914; 17:34, 1919; 18:31, 1920; 23:24, 1925; 26:26, 1928.

32. Derby, H.: Tr. Am. Ophth. Soc. 4:33, 1885.

he confessed that he did not know whether he had done so in other cases. E. Williams, of Chicago, said, "We have all heard that an open confession is good. Some time ago I perforated the sclerotic."

In 1885 Dr. Dennett reported on his invention of the first electric ophthalmoscope.³³

Dr. Knapp in 1887 made a report on 1,000 successive cases of extraction of cataract. Dr. Risley's study of progressive myopia, based on a record of 200,000 cases of opticians, and apparently showing a steady decline in percentage and degree, was made in 1894.³⁴

This is not the time when the many contributions of de Schweinitz (whose death all deplore) should be recounted, but mention must be made of his experimental studies on the toxic amblyopias³⁵ and of his masterly clinical studies of the relation of the ocular conditions to those of the organism, studies which showed the value and the wisdom of complete general examinations when combined with equally careful examinations of the visual organs.

It is somewhat surprising that the two most important single contributions to American ophthalmology, the causation of various nervous symptoms by errors of refraction and by ocular motor disturbances, were not presented before the society, though made by 2 members of the society. These members were William Thomson, working in conjunction with Weir Mitchell, who published the former study, and George Stevens, who published the latter. The fact that the first study was not presented before the society can probably be explained by its special importance from the point of view of neurologists. The facts concerning Stevens' contribution are more obscure.

The American Ophthalmological Society is a national organization. It is of interest then to inquire as to the geographic distribution of the members. In 1871 the 52 members resided in 10 states; one-half of them resided in New York, 9 in Massachusetts, 6 in Pennsylvania and the remaining 11 were scattered. In 1903 the 164 members resided in 23 states. Of these, 53, or 32.5 per cent, resided in New York; 28, or 17 per cent, in Pennsylvania; 21, or 13 per cent, in Massachusetts, and the remaining 62 were scattered in 20 states. In 1938 the 183 members were residing in 31 states and countries. Thirty-six, or 20 per cent, lived in New York; 26, or 14 per cent, in Pennsylvania; 16, or 9 per cent, in Massachusetts; 15, or 8 per cent, in Missouri; 11, or 6 per cent, in Illinois, and the remaining 79, scattered in 25 states and countries.

It will be seen that with the years and the increasing membership distribution of members has widened extensively and the relative preponderance of New York members has greatly diminished.

33. Dennett, W. S.: *Tr. Am. Ophth. Soc.* 4:156, 1885.

34. Risley, S. D.: *Tr. Am. Ophth. Soc.* 7:168, 1894.

35. de Schweinitz, G. E.: *Tr. Am. Ophth. Soc.* 6:23, 1891; 7:405, 1895.

I am reminded that in 1896 Dr. Jackson found that " $\frac{5}{6}$ of the membership of the A. O. S. was drawn from the Atlantic Coast States which contained less than $\frac{2}{5}$ of the population of the country."³⁶

I have been much interested in the number of contributions which members have made of scientific character and shall give a brief summary of those made by members who have passed away (table 2). There are 223 members in this group, which does not include the 45 who have already been considered as the founders. I have divided them into four groups: (1) those who did not present any papers, (2) those who presented a few (1 to 5), (3) those who presented a moderate number (6 to 11) and (4) those who read many papers.

In group 4 24 members read an average of 23 papers each, and of these 16 read less than the average and 8 presented a great number. The names of the 8 with the number of papers presented follow: Charles J. Kipp, 24; Samuel D. Risley, 26; Lucien Howe, 33; Charles A.

TABLE 2.—*Summary of Data Concerning Contributions of Deceased Members*

	Number of Members in Each Group	Average Number of Years of Membership	Average Number of Papers Presented	Total Number of Papers
Group 1.....	80	9	0	
Group 2.....	87	23½	2½	185
Group 3.....	32	35	8½	266
Group 4.....	24	38	23	549

Oliver, 36; George C. Harlan, 39; S. Theobald, 42; George E. de Schweinitz, 46, and Charles S. Bull, 49. They were all members during long periods. But it must not be thought that those who did not read papers or who read but few were short lived. Among the 80 who did not read papers there are 33 who were members more than twenty years, and 5 enjoyed between forty and fifty-six years of membership! Amazing silence!

I refrain from giving any statistics of the living members, 180 in number;³⁷ some still have time to improve their score! But it may interest the younger members, and perhaps encourage them, to learn that there are 11 living members who were elected forty or more years ago, 6 of whom are in the list of emeritus members; that there are 30 who have been members between thirty and forty years, 3 of whom are in the emeritus list, and that it is Dr. Jackson who in this respect, as well as in many others, heads the list, being a member for fifty-four years!

36. Jackson, E.: Ophth. Lit. 5:33 (March) 1915.

37. It is well to remember that the population of the United States increased vastly during this period of seventy-five years. The population in 1860 was 31,443,321, and in 1930 it was 122,775,046.

In giving these figures I trust that no one will think them a measure of the worth of the individual members, for they are not necessarily more of a gage than the weight of books would be of their value. Nor have the members contributed only through the papers presented. As officers, as members of the council and of the committees, in discussion of papers read and in the still more personal discussions during meetings and outside the meeting hall, they have given aid and guidance to each other, encouragement, incentive and inspiration.

It would be impossible to ascertain the entire value of the activities and the benefits of such an organization as the American Ophthalmological Society to its members and to the medical profession at large.

The amazingly long periods of membership of the great number of members is an index of their estimate of its worth to them and of their attachment. (I mention this with some hesitation fearing that it may lead to other than favorable criticism.)

TABLE 3.—*Incidence of Subjects Considered (in Percentage) According to Periods*

Subject	1864-1874	1896-1906	1929-1937
Visual tests of all forms.....	15.0	13.33	13.0
Anatomic and physiologic studies.....	7.0	3.75	5.7
New instruments (ophthalmic, etc.).....	9.0	7.5	6.68
External diseases, including iris and lens.....	17.0	19.4	15.25
Deeper ocular diseases.....	11.0	10.0	13.0
Ocular neoplasms.....	9.6	16.6	8.5
General organic and other diseases in their bearing on ocular condition.....	8.33	13.0	18.0
Operations, excluding cataract operations.....	7.6	10.0	6.6
Cataract operations.....	8.33	3.75	3.8
Experimental research problems.....	0	0.3	8.0

Twenty years ago Dr. Jackson ³⁸ wrote:

From the beginning its (American Ophthalmological Society) Transactions have reflected the interest of American ophthalmologists in the preponderance of short papers offering practical suggestions, or placing on record the chief features of striking cases.

A special committee reported in 1900 on "Supplementary Reports on Cases Previously Presented." ³⁹

In order to determine the special interests and the change of interest during the course of three quarters of a century, the subjects discussed during three ten year periods, 1864-1874 inclusive, 1897-1906 inclusive and 1929-1937 inclusive, were compared. During the first period 156 topics were discussed; during the second period, 294, and during the third, 315. The percentage of times certain topics were discussed in each period is presented in table 3.

38. Jackson, E.: Ophth. Lit. 5:1 (Jan.) 1915.

39. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 9:13, 1900.

It will be seen in this table that the subject of visual tests, which stood high in the first period, sank to the third rank in the last, while the subject of the relation of the diseases of the eye to general diseases and diseases of other organs, which occupied the sixth or seventh rank in the first period, has risen into the first rank in the last, having risen from 8.33 per cent in the first to 13 per cent in the second and 18 per cent in the third period.

The subject of cataract operations, which is represented by an incidence of 8.33 per cent in the first period, has fallen to something less than 4 per cent in the last. There is an element of surprise that neoplasms have held so prominent a position in the programs throughout

TABLE 4.—*First Honorary Members*

Name	City	Year of Election	Year of Death
Bethune, G. A.	Boston	1864	1896
Hays, Isaac.....	Philadelphia	1864	1879
Hooper, R. W.	Boston	1864	1885
Jeffries, John.....	Boston	1864	1876
Reynolds, E.	Boston	1864	1881
Wilkes, George.....	New York	1864	1876
Schweigger, C.	Berlin	1866	1906

TABLE 5.—*Additional Honorary Members*

Name	City	Year They Entered Active Membership	Year They Became Honorary Members	Year of Death
Garmalt, W. H.	New York	1864	1928	1929
Green, John.....	St. Louis	1866	1908	1913
Jeffries, B. Joy.....	Boston	1864	1913	1916
Knapp, Herman.....	New York	1869	1910	1911
Prout, J. S.	New York	1868	1911	1928
Sprague, Francis B.	Boston	1864	1901	1921
Holmes, E. L.	Chicago	1866	1898	1900

the history of the society. The most remarkable development is that experimental studies increased from 0.3 per cent (4 articles) in the middle period to 8 per cent (25) in the last period.

In its earliest years the society elected a small body of honorary members. These are presented, with the year of their election and the year of their death, in table 4. Since the selection of this group there have been additions, Dr. Harvey Cushing in 1938 and others who had long been active members, as shown in table 5.

In 1921 the society established an emeritus membership to relieve members who had served for many years from the obligations of active membership while retaining its privileges. The list is presented in table 6.

Finally, it is pleasant to recall the names of such whom the society has had as its eminent guests to deliver notable addresses or otherwise participate in meetings: Major Smith, 1908 and 1921; Professor Fuchs, 1911; Professor Morax, 1922; Dr. Magitot, 1922; Mr. Clegg, of Manchester, 1922; Mr. Basil Graves, 1925; Dr. Harvey Cushing, 1928; Mr. Treacher Collins, 1922; and Mr. George McKay, 1922.

The society in 1919 was the recipient of a fund from its then president, Lucien Howe, which was to furnish the Howe Medal to be awarded for

TABLE 6.—*Emeritus Members of the Society*

Name	State	Year They Entered Active Membership	Year They Became Emeritus Members	Year of Death
Deceased Emeritus Members				
Adams, A. E.	New York	1896	1923	1937
Bradford, H. W.	New Hampshire	1881	1923	1928
Oallan, Peter A.	Oregon	1876	1928	1932
Capron, F. P.	Rhode Island	1885	1931	1936
Culver, O. M.	New York	1890	1928	1938
Cutler, C. W.	New York	1903	1930	1935
Dixon, Lewis S.	Massachusetts	1874	1922	1924
Hansell, H. F.	Pennsylvania	1887	1928	1935
Holden, W. A.	New York	1896	1931	1937
Holt, E. E.	Maine	1883	1923	1932
Kollock, O. W.	South Carolina	1883	1930	1932
Lippincott, J. A.	Nice, France	1883	1933	1938
Maxson, S. C.	New York	1905	1921	1935
Minor, J. L.	Tennessee	1883	1929	1938
Pooley, T. R.	New York	1871	1921	1926
Quackenbos, A.	Massachusetts	1901	1930	1934
Randall, B. A.	Pennsylvania	1885	1925	1932
Reik, H. O.	New Jersey	1899	1922	1938
Ring, H. W.	Connecticut	1895	1922	1938
Sattler, Robert.	Ohio	1880	1938	1938
Spaulding, J. A.	Maine	1878	1921	1938
Theobald, S.	Maryland	1880	1923	1931
Wheelock, E.	New York	1891	1922	1930
Living Emeritus Members				
Curdy, R. J.	Missouri	1906	1938	
Finlay, O. E.	Cuba	1927	1938	
Jack, E. E.	Massachusetts	1895	1936	
Koller, Carl.	New York	1889	1933	
Lovell, D. B.	Massachusetts	1899	1938	
Marlow, F. W.	New York	1891	1836	
Shannon, J. R.	Virginia	1905	1936	
Weeks, J. E.	Oregon	1893	1935	
Wescott, C. D.	Illinois	1900	1935	
White, J. A.	Virginia	1892	1931	
Williams, E. R.	Massachusetts	1912	1938	

outstanding merit in ophthalmology. It was awarded to Carl Koller,⁴⁰ in 1922, to Alexander Duane in 1923, to Edward Jackson in 1926, to F. H. Verhoeff in 1932, to George E. de Schweinitz in 1934, to Arnold Knapp in 1937 and to the following foreign ophthalmologists: Ernst Fuchs in 1924, Priestly Smith in 1927, Theodor Axenfeld in 1929 and Sir John Herbert Parsons in 1936.

40. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 12:27, 1909. A committee was appointed to celebrate the twenty-fifth anniversary of Dr. Koller's discovery of cocaine as an anesthetic.

I should have liked to supplement this account with a full list of all past members and with short sketches of the more notable personalities, for the *Transactions* are very defective. I have succeeded in securing the life dates of most, the periods of their membership, the years of their scientific contributions to the society and such references as I could obtain to biographic sketches besides such as are found in the *Transactions*. These data are now arranged alphabetically and will be placed in the archives of the society, with the hope that such gaps as still exist will be filled before all memories pass.

In this hurried sketch I have traversed a period of three quarters of a century, when the society was founded, from the beginning of the specialization of ophthalmology and the weaning of ophthalmic practice from the surgeons, through the period when the students sought their training in European centers under great scholars and teachers. One finds that ophthalmology has reached a high development in this country, holding an important position in the medical schools; there are ample numbers of well equipped special hospitals and ophthalmic wards in general hospitals and a vast army of practitioners with good special training. This number is difficult to ascertain, but some indication is furnished by the fact that the Board of Ophthalmology has given certificates to 1,642, and the Section of Ophthalmology of the American Medical Association in 1938 had no less than 1,365 members. But it is not only in numbers that the development has taken place. The scientific literary output and the quality of the special journals and of the ophthalmologic works produced in this country have risen into the highest ranks. In recent years there have been established university departments, institutes and laboratories, providing the opportunity for the study of physiologic and pathologic problems. These require not only special laboratory equipment but, still more, that equipment which special training affords as well as that cooperation and help which scientific institutions with cognate departments and their investigators supply.

Some years ago Dr. de Schweinitz in a report dealing with questions of membership gave expression to the view generally held:

This Society is recognized as the representative gathering of ophthalmologists in the United States and Canada, and the Committee hopes that it may continue to hold this position, but it has very distinct rivals in the field. . . .⁴¹

It is clear that the line of progress in ophthalmology will be in the fundamental studies and researches just indicated, and these will be carried on by a growing body of men who are not practitioners of ophthalmology but biologic scientists. It is of utmost importance that

41. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 13:272, 1913.

they should be in close touch with the ophthalmologists and that the ophthalmologists should be deeply interested in the problems which concern both.

This society should, as leader, find a way of bringing into its body such scientific investigators who are devoting themselves to these fundamental problems of ophthalmology, problems which lie beyond the realm of clinical investigation.

It will be through the understanding and the cooperation of both the clinical and the experimental investigators of ophthalmologic problems, in their approach and in their mutual bearings and significance, that one may hope for great progress in ophthalmology.

And this is in accordance with the purpose of the society expressed in its first meeting seventy-five years ago and sustained throughout these years "the advancement of Ophthalmic Science and Art."

EXOPHTHALMOS, WITHOUT PULSATION, DUE TO ARTERIOVENOUS ANEURYSM

REPORT OF CASE

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Exophthalmos, which means protrusion of the eyeball from the socket, is an interesting condition for two reasons, namely, the great variety of lesions which produce it and the serious consequences it may cause to sight or life. The position of the eyeball in its socket varies in many persons and is dependent on the amount of fat present in the orbit, the configuration of the face and the size of the globe, highly myopic or buphthalmic eyes being apparently proptosed because of their large size. The average position of the eyeball in the orbit is such that if a straight edge is applied to the upper and lower margins of the orbit in a vertical direction, the cornea will be just posterior to it.

In cases of mild unilateral exophthalmos it is often difficult to be sure that the true exophthalmos is present. In these cases Bergin¹ has the patient seated, with the surgeon standing behind and looking down the patient's face from above; the patient is directed to raise his eyes gradually from the floor until the corneas become visible. The relative position of each cornea to the orbital margin then denotes the presence or absence of exophthalmos, provided the eyes are of the same size. For the accurate estimation of the amount of exophthalmos present, an exophthalmometer should be used.

I shall not attempt to list the many causes of unilateral exophthalmos in this paper. They may be found in Spaeth's² recent article on the subject.

REPORT OF A CASE

Mrs. E. H., a housewife 65 years of age, was admitted to the surgical service at the Abington Memorial Hospital on Aug. 3, 1938, complaining of a slight headache, a buzzing sensation in the left orbital region and throbbing pain and rapid loss of vision in the left eye.

Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, March 16, 1939.

1. Bergin, W. M.: Unilateral Exophthalmus, *Guy's Hosp. Rep.* **63**:245, 1909.

2. Spaeth, E. B.: Pathogenesis of Unilateral Exophthalmos, *Arch. Ophth.* **18**:107 (July) 1937.

She had had scarlet fever and diphtheria during childhood. She stated that three years before the present admission she was unable to talk for three weeks.

On July 29 the patient first noticed a buzzing sensation in the left orbital region, which on July 31 suddenly developed into a throbbing pain. She also noticed that the vision in the left eye was failing rapidly. On August 3 the buzzing sensation and throbbing pain were still present, and she became totally blind. The eyelids, conjunctiva and sclera of the left eye became greatly edematous, ecchymotic and of a purple discoloration. During the onset of the symptoms the vision of the right eye was not affected. The patient stated that previous to the present illness vision in each eye, with correction, had been good. She did not know what could have caused the condition and stated that there had not been any recent or past injury or fall. She said that she had not been feeling so well for the past twenty years because of heart trouble. She found that her blood pressure was elevated on a visit to her family physician two weeks before her admission.

The patient has one son and one daughter, both of whom are living and well at the time of writing. Her father died from a stroke and her mother of heart disease.

Physical examination revealed a quiet, cooperative patient in a semisitting position, suffering with a moderate amount of dyspnea. On her admission external ocular examination revealed a normal right eye and ophthalmoscopic examination showed the media to be clear and the disk of good color and well defined. The arteries were reduced in caliber, and the veins were slightly overfull and dark. There were marked swelling of the lid of the left eye, ecchymosis of the conjunctiva and subconjunctival tissues and marked proptosis. There was a marked increase in the intraocular tension. The pupil was semidilated and immobile to light but reacted consensually with the other eye. Examination of the left fundus showed the optic disk to be dusky in appearance, the nasal borders being barely visible, and the remaining margin to be obscured by swelling. The veins were dark and full. The arteries were thin and dark and pulsated. No hemorrhages or exudates were seen. A bruit was heard, systolic in time, over the orbital and temporal regions. There was complete immobility of all extraocular muscles. The heart rate was 120 beats per minute; the beats were very irregular due to auricular fibrillation. The blood pressure was 190 systolic and 108 diastolic. The temperature was 99.4 F. Otherwise the results of the physical examination were negative.

Laboratory examination showed a hemoglobin content of 110 per cent on two occasions. There were slightly over 5,000,000 red blood cells and 13,100 white cells; the differential count was normal. The Wassermann and Kahn tests of the blood were negative. The blood sugar and blood urea nitrogen were normal. The urine showed from a faint trace to a cloud of albumin, clumps of white blood cells and a few hyaline casts.

Roentgen examination did not reveal any evidence of intracranial disease. There were no shadows identified that were suggestive of atheromatous blood vessels. No areas of bone erosion were detected. An electrocardiogram showed auricular fibrillation with an average ventricular rate of 120, abnormalities of the T wave and myocardial damage.

On August 4 (the day after admission), the patient was given digitalis leaves, $1\frac{1}{2}$ grains (0.097 Gm.) daily. This dose was continued throughout her stay in the hospital and was found to be the correct dose to keep her heart compensating. Also on this day a course of manual compression of the left common carotid artery was instituted with the idea of later ligating the artery. In the beginning

the artery was compressed for five minutes every three hours, and this was gradually increased, until by August 11 it was possible to compress the artery for thirty minutes every two hours without symptoms developing. At this time the left eye showed slightly less exophthalmos. The cornea was slightly desquamated. The retina was markedly edematous, and the margins of the disk were almost obscured. The veins were dark, and there were many serous and sanguineous exudates. The intraocular pressure was above 95 (the McLean tenometer). The patient no longer heard the bruit, but it was plainly heard with the stethoscope.

At neurologic consultation on August 5 the patient was found to have most of the cardinal signs of an arteriovenous aneurysm into the left cavernous sinus. There were no neurologic signs referable to other parts of the body. Daily compression over the common carotid artery was advised rather than operation at that time.

On August 8 (five days after admission), peripheral paralysis of the left facial nerve developed which the neurologist thought was due to refrigeration as a result of the use of continuous wet dressings on the eye; it might have been due, secondarily, to edema in the soft structures along the base of the skull. There was no weakness of the extremities or impairment of hearing. There was no edema over the mastoid area on the left side. The left eye showed complete external ophthalmoplegia. The cornea was slightly clearer; the margins of the disk were blurred by hemorrhages which extended into the adjacent retina. There was some edema around the posterior pole; the veins were almost completely blocked, and the arteries were hardly seen. There was a suggestion of a beginning cherry red spot in the fovea. The bruit had completely disappeared.

On August 11 the left eye showed more marked conjunctival swelling and ecchymosis, and the palpebral fissure was nearly closed with the overhanging conjunctiva. The cornea was desquamated in the inferior exposed area. The paralysis of the facial nerve was more marked. The right eye remained normal. Examination on August 28 showed the swelling of the lids to be less and the exposure keratitis to be in the receding stage; there was an exudate in the anterior chamber beneath the previous ulcer. With the ophthalmoscope, a red reflex was barely obtained, and no fundus details could be made out.

On September 10, the patient was transferred to the ophthalmic service of Dr. J. Frederick Herbert. At this time the swelling of the lids was slight, and the keratitis was practically healed. Examination on September 24 showed that the condition had continued to improve, although there was no movement of any extraocular muscles and no fundus details could be seen.

The patient was discharged on October 5. By this time the exophthalmos and chemosis had gradually subsided. The hypoon was nearly absorbed, and the paralysis of the facial nerve was somewhat less. Intraocular tension was still markedly increased, and there was no vision present in the left eye. A red reflex was seen with the ophthalmoscope, but no fundus detail could be made out. The patient was only able to abduct the eyeball slightly, all other muscles remaining in a state of immobility.

The patient was again seen on November 9, and the examination revealed practically the same findings as on the date of discharge.

The patient was again seen on March 10, 1939, for a follow-up examination and for refraction of the right eye. This examination revealed a most interesting finding, viz., the return of the bruit after its absence for seven months. The bruit had been noticed by the patient for two weeks but was not particularly annoying to her. With the stethoscope the bruit was heard only over the eyeball and was

practically continuous, while seven months previously it was systolic in time and was heard over both the orbital and the temporal region. The remaining examination of the left eye gave results essentially similar to those found at the time of the patient's discharge from the hospital, Oct. 5, 1938.

At the time of writing the patient feels generally quite well, although she has a marked auricular fibrillation, with a pulse rate of 90 and a blood pressure of 180 systolic and 90 diastolic. On refraction she accepted for the right eye a $+2.75$ sph. with a $+0.50$ cyl., axis 180, which gave her vision of 20/20—3, and with the addition of a $+2.75$ sph. she was able to read Jaeger's test type no. 2. The fundus of the right eye showed moderate retinal arteriosclerosis; otherwise it was normal.

The only explanation I can give for the return of the bruit after its absence for seven months is that its disappearance was caused by a thrombus forming in the aneurysm, which later became recanalized so that the blood from the internal carotid artery again flowed directly into the cavernous sinus, thus causing the bruit.

Although I was never able to demonstrate any pulsation in the left eye in this case, I feel that a diagnosis of an aneurysm of the internal carotid artery into the cavernous sinus is justifiable.

REVIEW OF THE LITERATURE

In 1805 Travers described the first case of pulsating exophthalmos. Since this time much has been written concerning the subject. In 1907 de Schweinitz and Holloway³ reported 69 cases. In 54 the exophthalmos was due to trauma; in 13 it was due to spontaneous causes, and in 1 it was the result of a tumor; the cause in the remaining case was not given. The traumatic causes were falls or blows on the head in 37 cases, while in most of the remaining cases they consisted of gunshot wounds and puncture and penetrating wounds of the head. The average age of the patients with exophthalmos due to trauma was 30 years, while that of the patients with exophthalmos due to spontaneous causes was 42 years. The vision in most of these patients was markedly reduced, but in 13 eyes it remained normal. The cornea was affected in 8 cases, there being 4 cases of keratitis neuroparalytica and 4 cases of ulcer of the cornea. The involvement of the extraocular muscles varied from no involvement in 10 cases to complete external ophthalmoplegia in 5 cases. The most frequent muscle affected was the external rectus muscle. Optic neuritis, atrophy of the optic nerve, retinal hemorrhages and macular changes were frequently reported. In 20 per cent of the cases some degeneration of the optic nerve was found. In 9 cases the hearing was impaired or lost, while in 1 case taste was impaired and in another smell was markedly impaired. The ophthalmic branch of

3. de Schweinitz, G. E., and Holloway, T. B.: Concerning Pulsating Exophthalmos, *Tr. Am. College Physicians* 29:79, 1907.

the fifth nerve was involved in 5 cases, while involvement of the facial nerve was rare. Pulsation was present in nearly all the cases in this series, and bruit was present in all but 9. The bruit is most often systolic in time but may be continuous. It is often distressing to the patient and most often can be controlled by pressure over the carotid artery on the same side as the lesion, although at times pressure has to be made over both common carotid arteries.

Jack and Verhoeff⁴ reviewed the literature in 1908 and reported the results in 27 cases of pulsating exophthalmos in which autopsy was performed. They concluded that exophthalmos with vascular lesions is more often caused by disease behind the orbital cavity rather than in the orbital cavity.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis as to the exact lesion present is extremely difficult. Rivington⁵ gave the following hints as to the location of the lesion: (a) The sudden onset with pain and loud noise points to the rupture of an aneurysm or to its sudden formation. (b) The presence of paralysis of the orbital nerves points to an aneurysm or to an aneurysm which has burst, its clotted blood pressing on the cavernous sinus with its contained nerves. (c) Complete loss of vision at the outset and failure to recover it after ligation or digital compression would favor the supposition of an aneurysm of some other artery than the internal carotid artery. (d) The continuity of the bruit points to an arteriovenous aneurysm, and an interrupted bruit, to a true aneurysm.

TREATMENT

Many methods and theories of treatment of arteriovenous aneurysm in this location have been proposed. In Ruchlin's⁶ series of 68 cases in which digital compression was employed, cure was obtained in 15, while treatment resulted in failure in 52. The best results were obtained in the cases in which the exophthalmos was of spontaneous origin. Dorrance and Loudenslager⁷ expressed the belief that ligation of the internal carotid artery is contraindicated because of the greater number of postoperative complications noted. They stated that ligation of the common carotid artery is the most satisfactory treatment.

4. Jack, E. E., and Verhoeff, F. H.: A Case of Pulsating Exophthalmus: Ligation of the Common Carotid; Death; Pathological Report with Specimen, *Tr. Am. Ophth. Soc.* **11**:439, 1906-1908.

5. Rivington, W.: A Case of Pulsating Tumor of the Left Orbit, *Med.-Chir. Tr.* **58**:183, 1875.

6. Ruchlin, H.: Zur Kasuistik des doppelseitigen pulsierenden Exophthalmos, *Inaug. Dissert.*, Tübingen, 1902; cited by Jack and Verhoeff.⁴

7. Dorrance, G. M., and Loudenslager, P. E., cited by Bothe, F. A.: Treatment of Pulsating Exophthalmos, *Internat. Clin.* **1**:195, 1938.

In a reported series of 69 cases in which ligation of the internal carotid artery was done they found hemiplegia occurring 13 times; it was permanent in 4 cases, while in 3 other cases the recovery was slow, and in the remaining 6 cases the hemiplegia was only temporary. In a series of 82 cases in which the common carotid artery was ligated, hemiplegia occurred only 6 times; it was not permanent in any of the cases and was more than transient in only 1 case. From the review of the literature, I believe that ligation of the common carotid artery is the safest and most satisfactory procedure.

CONCLUSIONS

A rapidly developing pulsating exophthalmos, a bruit which is usually heard best over the orbital and temporal regions and rapidly failing vision, often resulting in complete blindness, are the cardinal signs of an aneurysm between the internal carotid artery and the cavernous sinus. In the case reported here all of the cardinal signs were present except pulsation, which is rarely absent in an aneurysm of the internal carotid artery and the cavernous sinus.

The condition in this case was severe, as evidenced by an extreme degree of exophthalmos, complete external ophthalmoplegia, acute glaucoma, an ulcer of the cornea, complete peripheral paralysis of the facial nerve and complete blindness.

As to the treatment in these cases, I believe ligation of the common carotid artery of the affected side, with subsequent ligation of several branches of the external carotid artery of the same side if necessary, to be the safest and most satisfactory form of treatment.

Dr. J. Frederick Herbert gave me permission to report this case.

AN INVESTIGATION OF EXPERIMENTAL CATARACTS IN THE ALBINO RAT

CLINICAL IMPLICATIONS

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For years it has been the aim of research investigators to find a procedure by which cataract might be developed experimentally in laboratory animals. This achievement they believed would create a better understanding of the changes observed in the ocular tissue of man leading to the formation of mature cataract. The contributions to this subject have been legion and the interpretation of the observations just as numerous. Notwithstanding all the accumulated literature and clinical and laboratory data, the causative factors of cataract formation remain an enigma.

It is known that lenticular changes may be present at birth or have their inception at puberty, progress at 40 and develop at 60, but not all elderly persons have mature cataracts. The incidence of cataract is great enough to warrant further investigation of the subject. It is conceded that no single factor is responsible for cataract formation in man. The present remedial methods are not altogether satisfactory in every instance, if at all. Most ophthalmologists will agree that medical treatment thus far advocated for the repair of already denatured lens tissue is unsatisfactory and that surgical intervention by competent operators offers the better solution to the problem. However, surgical treatment has its shortcomings, and there are some persons who would rather be blind than be operated on for cataract.

It seems reasonable that efforts should be made to prevent the formation of cataract. The question "Will it be possible to combat cataract by studying the chemistry of the lenticular changes?" has been raised by certain ophthalmologists who believe that each type of senile

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cataract is independently inherited. They are of the impression that the prospect of success in this field is about equal to that of finding a chemical means of avoiding inherited grayness of the hair, senile or presenile. There is a still larger group who believe that the general run of cataracts is due to the inevitable changes accompanying, more or less, the senile changes in other parts of the body. The following laboratory observations are presented in answer to these opinions relative to the causation of cataract.

Recently it was demonstrated that if the albino rat was fed a diet deficient in vitamin G (riboflavin), after a period of time inflammatory changes would develop in the ocular tissue that would ultimately produce characteristic opacities in the lens. Although all the investigators did not produce cataract with this deficiency ration, the ocular pathologic process was found frequently enough by others so that it may be accepted that vitamin G (riboflavin) plays an important role in the maintenance of healthy ocular tissue in the rat. In addition, it was also noted that the general health and growth of the animal were interfered with when a diet deficient in vitamin G (riboflavin) was fed. In contrast to the aforementioned experience with a deficient diet, Mitchell¹ observed in the course of her experiments with high rations of sugar that in experimental animals on diets high in lactose lenticular changes developed which led to the formation of mature cataracts. These lenticular disturbances were corroborated by Yudkin and Arnold² and by Day.³ It was apparent from this experiment with high rations of lactose and other sugars that it was the galactose fraction of lactose which caused the change in the ocular tissue. As a result of this finding, Mitchell⁴ and Yudkin and Arnold⁵ produced similar lenticular disturbances in animals by feeding them a diet of 25 and 35 per cent galactose. The rats on this ration of galactose supplemented with adequate amounts of the vitamins showed normal growth and maintained good health during the entire experiment. It was concluded from these experiments that the cataract was probably due to some metabolic disturbance unrelated to a vitamin deficiency. A résumé of the experimental investigation of galactose cataracts in our laboratory will be presented.

1. Mitchell, H. S., and Dodge, W. M.: Cataract in Rats Fed on High Lactose Rations, *J. Nutrition* **9**:37, 1935.

2. Yudkin, A. M., and Arnold, C. H.: Cataracts Produced in Albino Rats on a Ration Containing a High Proportion of Lactose or Galactose, *Arch. Ophth.* **14**:960 (Dec.) 1935.

3. Day, P. L.: Blood Sugar in Rats Rendered Cataractous by Dietary Procedures, *J. Nutrition* **12**:395, 1936.

4. Mitchell, H. S.: Cataracts in Rats Fed on Galactose, *J. Nutrition* (supp.) **9**:14, 1935; *Proc. Soc. Exper. Biol. & Med.* **32**:971, 1935.

5. Yudkin, A. M., and Arnold, C. H.: Cataract Formation in Rats Fed on a Diet Containing Galactose, *Proc. Soc. Exper. Biol. & Med.* **32**:836, 1935.

EXPERIMENTAL INVESTIGATIONS

BY DR. YUDKIN AND DR. GEER

The animals used for this problem were albino rats obtained from one source, the Connecticut Agricultural Experiment Station. The colony was known to be free of cataracts, and the stock was a healthy one. In the experiments females were used, and the litters were divided so that mates were in the experimental and in the control group. The albino rats were placed on the diet one to two days after weaning and weighed 40 to 45 Gm.

The basic diet initially used in these experiments consisted of 70 per cent carbohydrate (corn starch and galactose in varying proportions), 15 per cent casein, 9 per cent hydrogenated oil, 4 per cent Osborne and Mendel's salt mixture and 2 per cent cod liver oil. This diet was supplemented daily by 0.5 Gm. of yeast powder placed on top of the food. It was found that in 25 rats maintained on a diet containing 35 per cent galactose cataractous changes consistently developed.

It was observed that at the end of the fourth to the seventh day slight engorgement of the ciliary blood vessels was present, the vascular bed of the iris was slightly more prominent and the lens showed peripheral changes. This area was then invaded by small somewhat raised tissue, which was labeled "blisters." The blisters increased in number, and the subcapsular layer of the lens was involved at the end of twelve days. At this stage the number of blisters diminished, but the central nuclear portion became hazy, so that at the end of twenty-four to thirty days fully developed cataracts could be observed by the technician. A more careful study of the tissue with a beam of light from an ophthalmoscope and a Beebe loupe revealed that the blisters were vacuoles laden with fluid distributed underneath the lens capsule. Attempts to see the altered tissue with the slit lamp were extremely unsatisfactory. As the experiment progressed, the lens sutures were invaded with fluid. This step could not be seen in all lenses. In the young animals the lesion seemed to involve the nuclear layers rapidly, for the surrounding tissue became cloudy after the tenth day and the nucleus on the eleventh or twelfth day. Macroscopically, however, it appeared that the nucleus became opaque before the surrounding tissue, but this observation was not correct, for with higher magnification the surrounding tissue of the nucleus was invaded first by changes and then the nucleus itself became opaque. After this stage the opacity spread into the outer layers of the lens, so that at the end of thirty days almost the entire lens was opaque in most of the animals on the ration containing 35 per cent galactose. During the period the animals maintained normal growth, appeared healthy and showed no other physical defects.

When the amount of galactose was decreased to 25 per cent definite opacities of the lens developed in only two thirds of the rats. With a further decrease of the galactose to 15 per cent, no definite ocular changes were produced in 10 rats maintained on this diet from three to seven months.

In order to study the effect of protein on the formation of galactose cataract, 10 rats were placed on a diet containing 35 per cent casein, 35 per cent galactose and 15 per cent corn starch. The other constituents of the diet were the same as in the initial experiments. Three control litter mates were given a diet containing 15 per cent casein, 35 per cent galactose and 35 per cent corn starch. Although the high protein may have slightly delayed the appearance of the ocular changes, there was no clearcut difference in the progression of type of change that occurred. The appearance of the initial change in the rats on the diet high in protein occurred on an average of five and two-tenths days except for 1 rat, which showed no such change, whereas the initial change in the control rats occurred on an average of three and seven-tenths days. Lenticular opacities were noted in all of the rats at the twelfth or thirteenth day. Mature cataract developed in one or in both eyes of 7 of the 10 rats on the diet high in protein and in 2 of the 3 control rats. Although opacities were present in the lenses of all the rats, the immature cataracts did not become mature with continuation of the diet for seven months.

In the previous work of this laboratory cataractous changes were not observed in the eyes of rats maintained on a diet containing 15 per cent galactose and 15 per cent protein. When the protein of the diet was decreased to 5 per cent and the galactose to 15 per cent, a marked effect on the formation of galactose cataract was found. In all 10 rats placed on a diet containing 15 per cent galactose, 5 per cent casein and 65 per cent corn starch, peripheral changes and opacities developed. In 4 rats the changes progressed to mature cataracts. The peripheral change appeared within from four to twelve days and the opacities from nine to twenty days. In 3 of the rats the cataracts matured in less than thirty days. The fourth cataract did not become mature until the fourth month.

The diet was altered by decreasing the galactose to 10 per cent with the protein at the low level of 5 per cent; control litter mates were fed a diet with 15 per cent galactose and 5 per cent casein. As was to be expected, 10 per cent galactose was found less effective than 15 per cent galactose in producing cataractous changes. Of 5 rats placed on the diet containing 10 per cent galactose, 3 showed only blister changes, although peripheral changes developed in 2 at fourteen and twenty-eight days, with definite lenticular opacities within one month. The control rats all showed peripheral changes, and lenticular opacities consistently developed.

Experiments with cystine were repeated because our previous observations were not in accord with those of other investigators. The diet containing 15 per cent galactose and only 5 per cent protein was used in the experiment with cystine, for it consistently produced ocular changes in weaning rats. Two different groups were placed on this regimen. To the food of one group 4 per cent cystine was added; to that of the other, 2 per cent cystine. The control litter mates were maintained on the same diet without the addition of any cystine. Thirteen rats were placed on the diet containing 4 per cent cystine, with 7 rats as controls. Although all but 1 of the control rats showed the initial peripheral change within two to twenty-four days, none of the rats given cystine exhibited this type of change. In many of these animals small pinpoint opacities were observed. It was characteristic of these opacities that they never increased appreciably in size and often disappeared entirely. As a result of the large amount of cystine, 1 of the experimental animals died at the end of a month. The other rats were maintained on the diet for periods of four to seven months.

When only 2 per cent cystine was added to the diet, the general condition of the animals was superior to that of the animals receiving larger quantities of cystine, and the ocular changes were greater. Five animals were placed on this diet with 4 control rats. Two of the 5 showed peripheral changes at eight and nine days; opaque plaques developed in 2, and all of them showed pinpoint opacities after one or two months on the experimental diet. After four months, however, only the blisters described previously could be detected. The 4 controls, on the other hand, showed lenticular opacities at the time they were killed. It may be noted from the aforementioned data that cystine in large amounts was efficacious in preventing the development of galactose cataract. Cystine in these amounts, especially 4 per cent cystine, had a definitely deleterious effect on the rat. Many of the rats did not increase normally in body weight. At autopsy some of the rats showed nutmeg livers with fatty infiltration.

The effect of large amounts of riboflavin on galactose cataract was found to be negative. These rats were fed the diet containing 15 per cent galactose and 5 per cent casein. As much as 4 mg. of riboflavin⁶ mixed with 1 cc. of olive oil was given orally by pipet to each rat six times a week. Although some of the rats were maintained on this regimen for as long as two months, no significant difference was noted between the ocular changes produced in the animals receiving riboflavin and those deprived of the vitamin. Mitchell and Cook⁷ reported similar results with riboflavin.

6. Riboflavin was made available through Merck and Company.

7. Mitchell, H. S., and Cook, G. M.: Effect of Riboflavin and Thiamin Chloride upon the Cataractogenic Action of Galactose, *Proc. Soc. Exper. Biol. & Med.* **39**:325, 1938.

In previous work of this laboratory⁸ dinitrophenol had been given both orally and by injection of a 2 per cent aqueous solution to albino rats. The diet used in these experiments contained 23 per cent corn starch, 31 per cent dextrose, 18 per cent casein, 15 per cent hydrogenated oil, 6 per cent roughage, 4 per cent Osborne and Mendel's salt mixture and 3 per cent cod liver oil. The maximum amount of dinitrophenol was given to 8 rats which were 2 months old at the start of the experiment. The food intake of these animals was limited to 6 Gm. per day. Oral administration of dinitrophenol was increased from 2 to 12 mg. daily over a period of ten months. The rats were excitable and irritable. Two were killed before the completion of the experiment because of their poor general condition, and no ocular changes were noted. Similarly, no ocular changes were noted in any of the rats given dinitrophenol.

An attempt was made to produce galactose cataract by the administration of dinitrophenol to rats receiving a diet of 15 per cent galactose and 15 per cent casein. Eight weaned rats were maintained for a period of five months on this diet. The dinitrophenol which was placed on top of the food daily was increased from 1.5 to 3.5 mg. No ocular changes were observed.

It was important to decide whether or not the intestinal tract was involved in the production of galactose cataract, so peritoneal injections of 30 per cent aqueous solutions of galactose were made. The first injections consisted of 1 cc., and the dose was increased to 4 cc. as the experiment progressed. Owing to the fact that large doses of the concentrated solutions temporarily paralyzed the hindlegs of the rat and often killed the animal, less galactose was given than would be obtained orally from a diet containing 15 per cent galactose. Control litter mates were placed on a diet containing 15 per cent galactose and 5 per cent casein. Three different diets were used in these experiments: (1) 15 per cent dextrose and 5 per cent casein to 5 rats, (2) 15 per cent dextrose and 15 per cent casein to 4 rats and (3) a stock calf meal diet to 8 rats. The other constituents of the dextrose diet were the same as those in the aforementioned galactose diets. The most marked ocular changes were observed in the animals on the diet low in protein. Four of these animals showed the initial peripheral change within seven to nine days; the fifth rat had a small lenticular opacity at the end of fifteen days. In an effort to increase the intake of galactose, 2 of these rats were given an injection of 1.5 cc. instead of 1 cc. At the end of a month the hindlegs had become permanently paralyzed. Although the injections were discontinued, their condition did not improve. These

8. Yudkin, A. M.: Diet and Vitamins in Relation to Cataracts, *Am. J. Ophth.* **21**:871, 1938.

animals died within a short time, and it was noted that lenticular opacities were present. The rats remaining on this experiment over a period of four months showed opacities. In some lenses the opacities seemed to disappear, but all of the rats showed blisters in the subcapsular tissue. Two of the 4 rats on the 15 per cent dextrose and 15 per cent casein showed peripheral changes; at the end of four months only blisters could be seen. The injections for the 8 rats on the stock calf meal diet were increased over a period of four months from 1 cc to 4 cc. In spite of the fact that mature cataracts developed in the control litter mates, these rats showed only blisters. These changes were not marked and could be seen in only 5 of the rats.

In this laboratory 14 rats in which mature cataracts had developed were placed on a normal diet without galactose. The galactose diet was withdrawn from five days to two weeks after the cataract had become mature. Seven of these animals were maintained on a stock diet from ten to twelve months. The clearing at the edges described by Mitchell and Cook⁹ was noted in these animals, sometimes as soon as a week after removal of the galactose from the ration. In no animal did the nuclear opacity disappear from the lens as described by other workers.

COMMENT

The ocular changes and cataract formation associated with deficiency of vitamin G (riboflavin) differs somewhat from that produced in the albino rat on a ration of 15 per cent or more galactose. Recently Eckardt and Johnson¹⁰ confirmed this observation by showing that corneal vascularization was the most consistent ocular change in the albino rat on a diet deficient in vitamin G and that cataracts were found in only a few of the animals used for the experiment. When riboflavin was added to the deficient diet, the vascularization of the cornea disappeared before any other ocular disturbance. In the experiments with galactose they found no corneal lesions, but the changes in the lens were consistent. Eckardt and Johnson also noted that a senile suture pattern developed in three weeks in rats fed a ration high in lactose. This lesion was accompanied by peripheral vacuoles as well as by club-shaped riders similar to those seen in coronary cataracts. The senile suture pattern was not observed in any of the rats on the lactose-free diet. These observations are similar to those made by Sasaki.¹¹ He also found

9. Mitchell, H. S., and Cook, G. M.: Galactose Cataracts in Rats: Factors Influencing Progressive and Regressive Changes, *Arch. Ophth.* **19**:22 (Jan.) 1938.

10. Eckardt, R. E., and Johnson, L. V.: Nutritional Cataract and Relation of Galactose to Appearance of Senile Suture Line in Rats, *Arch. Ophth.* **21**: 315 (Feb.) 1939.

11. Sasaki, T.: Untersuchungen der Linse, des Blutes und des Kammerwassers von Ratten bei Galaktoseverfütterung: Klinische Beobachtungen bei der Glukosekatarakt, *Arch. f. Ophth.* **138**:351, 1938.

opacities in the lens three to seven days after the diet was started; the opacities were situated around the sutures and were subsequently covered by subcapsular radial opacities. The American investigators, however, noted changes in the capsule and subcapsular tissue before the opacities appeared in the substance of the lens.

This clinical picture of cataract formation in the animal fed galactose has been confirmed in the main by Gifford and Bellows.¹² They reported that in the galactose cataract the earliest changes involve the cortical fibers near the equator, but growth of new fibers was rapid enough so that some relatively normal fibers were always present just beneath the capsule in this region. They found that the changes depended on the duration of the feeding of galactose. The lesions were similar in type but affected areas of varying extent. The lens fibers in the involved area lost their interfibrillar membranes and changed in staining properties, the normal homogeneous fibers being replaced by a granular mass of pink-stained material. In later stages this material was replaced by almost clear fluid, with fine blue-staining or pink-staining granules in suspension. The nuclei near the equator showed early degenerative changes, losing their oval form as the lens fibers became liquefied and becoming round and pale or in some cases small and dark.

Further work on this problem will no doubt lead to a better understanding of the formation of galactose cataract. This type of cataract can be produced slowly or rapidly, depending on the amounts of galactose used or by varying the amounts of protein and cystine in the diet. Geer and I have confirmed the observations of Mitchell and Cook⁹ and noted that a decreased amount of protein required a smaller level of galactose to produce cataracts. We cannot quite substantiate their observations that a high protein content retards noticeably the process of cataract formation. We agree with Bellows¹³ that cystine retards the formation of cataract in the animals fed galactose, but it requires a supplement of at least 4 per cent of cystine to perform this retardation. It was noted that the addition of 4 per cent cystine interfered with the metabolism of the animal and produced pathologic changes in some of the internal organs, particularly the liver.

Since Day and his co-workers¹⁴ showed evidence of preventing and even arresting cataracts in the albino rat by the injection of crystalline riboflavin, it seemed logical to try the chemical in other animals on

12. Gifford, S. R., and Bellows, J.: Histologic Changes in the Lens Produced by Galactose, *Arch. Ophth.* **21**:346 (Feb.) 1939.

13. Bellows, J. G.: Biochemistry of the Lens: IX. Influence of Vitamin C and Sulphydryls on the Production of Galactose Cataract, *Arch. Ophth.* **16**: 762 (Nov.) 1936.

14. Day, P. L.; Darby, W. J., and Cosgrove, K. W.: Arrest of Nutritional Cataract by Use of Riboflavin, *J. Nutrition* **15**:83, 1938.

cataract-forming diets. After several attempts, it was noted that the addition of large amounts of vitamin B complex or riboflavin to the diet did not interfere in any way with the progress or retardation of cataracts in the animals given galactose. This observation was also made by other investigators.⁷

In view of the numerous clinical reports that the administration of dinitrophenol to certain patients has been followed by the development of cataract, it was thought that a similar condition might be produced in the experimental animals. Arnold and I fed several groups of albino rats large doses of dinitrophenol without observing the slightest change in the lens.⁸ Many of the animals succumbed after having been on the diet two months. It was suggested that dinitrophenol be added to the diet of animals on a low galactose ration to see whether its presence in the diet would have any effect on the production of lenticular changes. The results were similar to those of Borley and Taintor,¹⁵ who reported that the addition of dinitrophenol to the cataract-producing diets did not alter the progress or the retardation of the cataract.

It is agreed by most investigators that the ingestion of galactose by the experimental animal increases the nonfermentable fraction of the blood sugar, and it is somewhat higher than that observed in the animals fed lactose. On the strength of this finding Mitchell and her co-workers¹⁶ concluded that galactose is the sugar responsible for the high sugar content of the blood and urine of rats fed lactose or galactose rations and must be the major etiologic factor in galactose cataracts. Sasaki¹⁷ made similar observations on the blood and urine and also noted that the sugar content of the aqueous humor is considerably increased. Yudkin and Arnold² injected various amounts of galactose in animals and found an increased content of the blood and urine sugar in some of them. It was noted that only early changes, such as congestion of the iris and blisters, were produced in the subcapsular tissue of the lens. It was difficult to maintain these animals on this regimen over a long period of time because of the paralysis of the hindlimbs and the poor growth and health of the animals. This experiment is important, for it shows that galactose must have some effect on the permeability of the lens capsule. This impression is held by most investigators, but the type of change, increased or decreased permeability of the lens capsule, is still undetermined.

15. Borley, W. E., and Taintor, M. L.: Influence of Dinitrophenol on the Production of Experimental Cataracts by Lactose, *Am. J. Ophth.* **21**:1091, 1938.

16. Mitchell, H. S.; Merriam, O. A., and Cook, G. M.: The Relation of Ingested Carbohydrate to the Type and Amount of Blood and Urine Sugar and to the Incidence of Cataract in Rats, *J. Nutrition* **13**:501, 1937.

17. Sasaki, T.: Untersuchungen der Linse, des Blutes und des Kammerwassers von Ratten bei Galaktoseverfütterung: Ueber die Linsenkapselpermeabilität und über den Zuckergehalt von Blut, Kammerwasser und Linse bei der Galaktose-Kataract, *Arch. f. Ophth.* **138**:365, 1938.

Bellows and Rosner¹⁸ observed that galactose decreased the permeability of the capsule of the fresh beef lens when it was subjected to a modified Friedenwald¹⁹ technic for determining the permeability of the capsule. On the strength of this finding, they concluded that the production of cataract in the animal fed galactose may be due to the decreased permeability of the lens capsule. It is rather interesting that Friedenwald²⁰ also showed that the permeability of the capsule of the lens decreases with age. If galactose decreases the permeability of the capsule, it is conceivable that this alteration may prevent sources of nutrition from reaching the lens and thus hasten death of the fibers of the lens, with a resulting cataract.

It would be ideal if the problem of cataract formation could be so easily solved, but there are many other factors besides the permeability of the lens capsule that must be considered. This report does not permit delving into the respiratory phase and nutrition of the normal and the pathologic lens. It is important, however, to mention the possibility of retarding the formation of the experimental cataract at various stages of its development by diet. Most investigators have noticed that if the galactose diet is changed to a normal diet before the nuclear layer becomes hazy, the visible changes in the lens, such as blisters, peripheral changes and subcapsular opacities, will frequently disappear and the lens become clinically transparent. Mitchell and Cook⁹ have observed that when what they called posterior opacities developed in an animal the change through the nuclear stage to that of complete opacity proceeded at a remarkably uniform rate. In all but a few instances the lenticular changes progressed to complete opacity irrespective of the diet utilized during this period. When the animal was shifted to a normal diet during this stage, the lenticular changes continued to completion, but at the end of three weeks they noticed a clearing of the cortex at the equator. This apparent regression continued until the dense opacity had receded to a nuclear area. In many eyes the dense central opacity became as small as a pinhead, with the peripheral zone entirely clear, and in rare instances this dense spot seemed to fade until it appeared like an early posterior opacity. The animals placed on a normal diet did not show as marked a retrogression. It is our belief that the opaque areas in the lens cannot be altered by normal diet to the extent that the lens regains its transparency. The changes, such as blisters, small

18. Bellows, J., and Rosner, L.: Biochemistry of the Lens: XI. Effect of Galactose on the Permeability of the Capsule of the Lens, *Arch. Ophth.* **20:80** (July) 1938.

19. Friedenwald, J. S.: Permeability of the Lens Capsule to Water, Dextrose, and Other Sugars, *Arch. Ophth.* **4:350** (Sept.) 1930.

20. Friedenwald, J. S.: Permeability of the Lens Capsule, with Special Reference to the Etiology of Senile Cataract, *Arch. Ophth.* **3:182** (Feb.) 1930.

discrete opacities in the outer layers of the lens and haziness of the tissue surrounding the nucleus before opacification takes place, can be repaired to the extent of the ability of the lens tissue to eliminate the accumulated fluid and of the tissue to regenerate. Further investigation is necessary before the final word can be written on the development of galactose cataract. Enough has been accomplished in this field so that clinical implications can be speculated.

CLINICAL IMPLICATIONS

BY DR. YUDKIN

It is extremely difficult to visualize the clinical picture of the formation of cataract in experimental animals if one is not acquainted with the appearance of the changes in the lens as they are observed by the laboratory investigator. My interpretation of the clinical manifestations is somewhat different from that of the laboratory investigators. They have described the early signs of ocular disturbance as blisters, peripheral changes and haziness; whereas the first changes are congestion of the circumcorneal area and fulness of the blood vessels of the iris followed by changes in the lens capsule. The subcapsular tissue becomes laden with vacuoles of fluid, and sutures of the lens are similarly invaded. The deeper layers are sprinkled with minute discrete opacities; the nuclear tissue then becomes opaque, and finally the whole lens becomes opaque within one month. When one realizes that the life span of a rat is short and every day of its existence is equivalent to ninety days of man; that the method used for producing the cataract is extreme, and that the development of the cataract in animals on a diet containing 50 or 35 per cent galactose is so rapid that one stage of cataract formation blends into another without giving an opportunity of differentiating the changes which take place, one may see some possible similarity between the experimental cataract and a type observed in man. To my knowledge, the only instance of that type of progress in the development of cataract is that associated with the ingestion of dinitrophenol or allied substances for the sole purpose of reducing weight. A résumé of the reports of dinitrophenol cataract would lead to speculation that its development is similar to that seen in the animals fed galactose.

The ophthalmologist who has been in practice for a long period in a community in which he is able to observe and study the same patients for many years can probably visualize the process which has been described in the development of galactose cataracts. Every practitioner has seen these stages in different patients. It would take the lifetime of an ophthalmologist to see the development of cataract in man as it is seen in the experimental animal. I therefore propose that if and when

vacuoles are seen with an ophthalmoscope in the capsular and sub-capsular tissue the patient should be considered as a potential subject for the development of lenticular changes.

It is surprising how many opacities are found in the lens of persons past 50 years of age if the pupil is dilated and the extreme periphery examined. The lenticular changes in these instances are frequently insignificant and in many cases remain stationary for years or advance so slowly that they may never interfere with vision. On the other hand, there are some cases in which the changes in the lens may go on to further opacification of the lens tissue if the health of the patient is not improved or the source of toxin removed. I do not subscribe to giving the patient at this time any information about cataract formation but deem it necessary to have a complete physical examination made after the family physician has been informed of the changes observed in the lens. He should be instructed to inquire about (1) the patient's personal hygiene, (2) his diet, (3) a history of gastrointestinal disturbances and (4) vascular decompensations and any other conditions that are necessary for the maintenance of good health in elderly persons.

Many ophthalmologists have been criticized because of their dietary regimen when it is employed at the stage when early lenticular changes are noted. It has been shown that as age advances and activities in general are curtailed there is correspondingly less need for food. There are also some persons who show some of the so-called degenerative diseases, such as arteriosclerosis, rheumatism and many others. Elderly persons require minerals, vitamins and proteins to maintain good health, even though the demand for the energy foods, carbohydrate and fat are greatly reduced. Many elderly persons curtail their diet at the expense of mineral and vitamin-containing foods and take a relatively high carbohydrate diet. Loss of teeth, development of idiosyncrasies to food or vague digestive disorders may cause the elderly person to drift into poor dietary habits. There is little doubt that many elderly persons could be restored to an active healthy happy existence by attention directed to their diets.

Some ophthalmologists have tried this course of treatment and observed no further change in the disturbance in the lens, and in some instances the condition became worse. It is not enough to tell the patient how to live, eat and pursue happiness; inquiry must be made as to whether the treatment is followed with the utmost amount of care. It has been my experience that some practitioners question the real value of this medical approach for the relief of early changes in the lens because they have observed that changes go on to maturity in spite of the best laid plans.

There is sufficient proof, however, that the patient is at least restored to good health if he is placed on a well balanced diet, and this is helpful

when operation for cataract is contemplated. The ophthalmologist should profit by the experience of the laboratory investigator and place the patient with incipient cataract on a well balanced diet supplemented by vitamin B complex and lemon juice and see what results can be obtained. I believe that it is important to be scientific, but one should not lose sight of the practical side of the question too.

CONCLUSION

The albino rat is susceptible to lenticular changes as a result of feeding lactose at high levels or galactose at lower levels. It is now possible to study the formation of opacities in the lens under carefully controlled conditions. It is apparent that a low protein ration hastens the development of the opacities of the lens and that a high protein ration may retard it; a high intake of cystine also tends to retard the pathologic change. Dinitrophenol given in large doses does not produce lenticular changes in animals, nor does it aid in the formation of cataract. Massive doses of riboflavin and liberal amounts of vitamin B complex in the form of brewers' yeast powder exert no protective action against the development of galactose cataract in rats. When a lens has become completely opaque (mature cataract) in the rat as a result of the aforementioned methods, no specific dietary factor or local medication has been found that will change the opaque lens to its former transparent state. It is possible, however, to check the early development of the lenticular changes by altering the diet. When early changes are observed in man he should be instructed (1) in personal hygiene, (2) to utilize a well balanced diet, (3) to correct, if possible, gastrointestinal dysfunctions and (4) to adjust vascular decompensations.

COURSE IN CERTAIN CASES OF ATROPHY OF THE OPTIC NERVE WITH CUPPING AND LOW TENSION

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In the cases reported here, in which there were cupping of the atrophic optic disk and low ocular tension, the changes in the visual fields were not common to glaucoma and remained stationary in their course for a long time. To study this phase, in which they also differ from the course in cases of the usual type of glaucoma, I endeavored to follow up the cases which were reported in an article on "Association of Sclerosis of the Cerebral Basal Vessels with Optic Atrophy and Cupping," presented before the American Ophthalmological Society in 1932, and I am now ready to report on the subsequent histories of those which are available and of several which have been added since that date, making 11 cases in all.

In this series a group of cases can be distinguished in which the changes in the visual field are altitudinal. The condition in such cases is frequently characterized by a stationary course.

REPORT OF CASES

CASE 1.—Del. S., aged 53, had been observed since July 15, 1930. On that day the vision was 20/20 in the right eye and 20/20—in the left eye. The tension was 31 in each eye. The fields presented an altitudinal hemianopia. Roentgen examination revealed calcification of the internal carotid arteries. The patient was last seen on July 18, 1938, with vision of 20/20—in each eye and tension of 26. The fields were unchanged. The patient has been using 1 per cent solution of pilocarpine hydrochloride.

CASE 2.—S. W. K., aged 71, was seen on March 9, 1937. In the right eye the vision was 20/50 and the tension 31. In the left eye vision was 20/20 and the tension 36. The fields showed an inferior hemianopia. A roentgenogram of the skull revealed calcification of the internal carotid arteries. On Oct. 18, 1938, the vision was unchanged, but there was a temporal increase in the inferior hemianopia. The tension measured 20 in the right eye and 26 in the left eye. In December 1938 the patient suffered from an attack of unconsciousness; the blood pressure was 200. This was followed by vertigo and a failure of sight. On May 29, 1939, the vision was 20/50—in the right eye and 20/30—in the left eye. The

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superotemporal defect in both fields had increased and had reached 50 degrees from the vertical meridian. The tension was 25 in the right eye and 36 in the left eye.

This is the first instance in which a definitely altitudinal defect did not remain stationary, and the increase in the field defect is interesting.

CASE 3.—V. M., aged 69, was seen on Jan. 28, 1930, with vision in the right eye of 20/20 and tension of 20. The field showed an altitudinal defect in the upper quadrant and an unusual cecocentral scotoma. A roentgenogram revealed calcification of the internal carotid and of the ophthalmic arteries. The left eye was blind from an old central choroiditis. The vision and fields remained stationary until 1936, when a gradual loss of vision began. The tip of the scotoma then gradually included more and more of the point of fixation, and on May 5, 1938, the vision in the right eye was reduced to 20/50. The tension was never above 20, and the patient used miotics irregularly. Death occurred suddenly in June 1938, from a "heart attack."

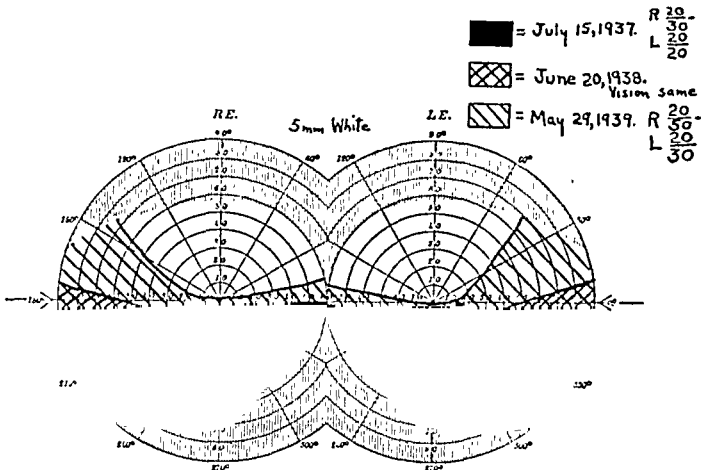


Chart 1 (case 2).—Visual fields of Mrs. S. K.

CASE 4.—Mrs. E. S., aged 63, was seen on April 24, 1930. The vision in the right eye was 20/20—, and the tension was 26. The vision in the left eye was 20/70, and the tension was 22. The fields showed irregular altitudinal defects. Roentgen examination revealed calcification of the posterior communicating arteries and in the optic foramen. The sight then failed, and a cataract developed in each eye, so that examination of the fields was impossible. The vision on Dec. 16, 1938, was reduced to perception of hand movements in each eye; the tension was 21 in the right eye and 18 in the left.

The patient's poor sight could not be entirely ascribed to the cataract but was also due to progress of the lesion of the optic nerve.

In the remaining cases reports irregular and not characteristic field defects are presented.

CASE 5.—Mrs. D. H., aged 63, was seen on April 17, 1931. The vision in the right eye was 20/30— and in the left eye 20/30. The tension in each eye was 18. The field of the right eye presented a concentric contraction, while in the left

eye the defect was principally down and in. Roentgen examination showed calcification of the internal carotid arteries and of the right ophthalmic artery.

The patient was last seen on Jan. 25, 1939, by her oculist, Dr. W. Blake Gibb, of Morristown, N. J., who reported that the vision in the right eye was 20/100 and in the left eye 20/30. The tension was 20 in the right eye and 18 in the left. At the time of writing the fields showed a concentric contraction.

CASE 6.—Mrs. E. B. S., aged 60, had on March 5, 1932, vision of 20/30 in the right eye and 20/20—in the left. The tension in each eye was 16. The fields showed an irregular altitudinal defect. A roentgenogram revealed calcified changes in the internal carotid arteries. Trephining was done by her physician, Dr. J. J. Wynne, of Louisville, on one eye in November 1934 and on the other in January 1935, because a contraction of the fields was observed.

On Jan. 22, 1939, the patient was examined by Dr. J. J. Wynne, who sent me the following report:

Vision in the right eye, which contained a cataract, was 20/200. Vision in the left eye was 20/20. The tension was soft in each eye. The left field showed no particular change; the right field could not be taken.

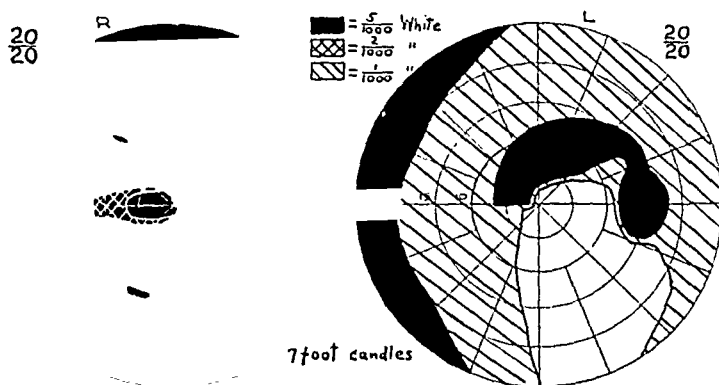


Chart 2 (case 8).—Visual fields of Mrs. C. B., taken on Sept. 19, 1938.

CASE 7.—C. F. R., aged 68, was seen on Jan. 3, 1925, with vision of 20/70 in the right eye and tension of 26. The left eye had vision of 20/200 and tension of 22. Each field showed an irregular nasal hemianopia. A roentgenogram revealed calcification of the internal carotid arteries. A nuclear cataract was present in each eye, and iridectomy was performed. The cataract in the right eye was then extracted in the capsule on April 16, 1929, and on Feb. 2, 1931, the vision was 20/70 in the right eye with tension of 12 and 2/200 in the left eye. On Jan. 28, 1939, the patient was seen by Dr. H. S. Niles, of Bridgeport, Conn., who sent me his findings, which showed that the patient was blind and the tension increased.

CASE 8.—C. B., aged 70, was seen on Sept. 26, 1938, through the courtesy of Dr. R. K. Lambert, of New York. Vision had been failing for an indefinite time, but with correcting glasses it still was 20/20 in the right eye and 20/20 in the left eye. Both optic disks were atrophic and depressed. The tension was never above 16 in the right eye and 18 in the left eye. The right field was much contracted, and two minute islets remain. The left field was interesting as it showed in addition to other changes an arcuate scotoma somewhat like that seen in cases of glaucoma.

CASE 9.—C. L., aged 75, was seen on Jan. 26, 1939, through the courtesy of Dr. Paul Chandler, of Boston. A diagnosis of glaucoma had been made, and an operation was considered. Vision in the right eye was 20/200 and in the left eye 20/20. The right field presented a nearly complete superior defect, and in the left field the defect occupied the upper and nasal quadrant and there was an atypical cecocentral scotoma approaching the arcuate variety. The right optic disk was atrophic and partially cupped; the left disk was pale, and the vessels were displaced nasally. The tension on repeated examinations was 21 in the right eye and 18 in the left eye. A roentgenogram revealed suggestive but no definite changes.

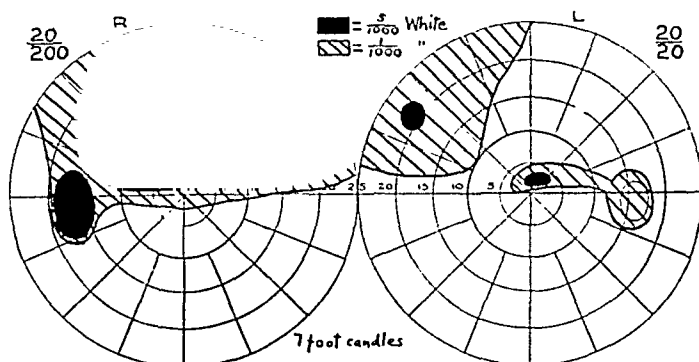


Chart 3 (case 9).—Visual fields of C. L., taken on Jan. 27, 1939.

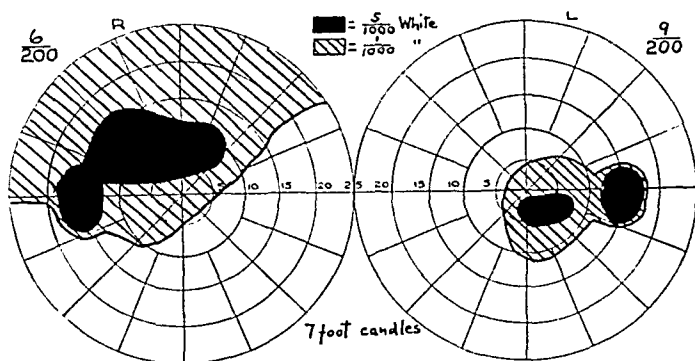


Chart 4 (case 10).—Visual fields of G. C., taken on Feb. 14, 1939.

In cases 8 and 9 the changes in the visual fields in part suggested glaucoma, but the other symptoms were not characteristic. These cases bear out Traquair's statement that a definite diagnosis cannot be made from the changes in the visual field alone.

CASE 10.—G. C., aged 74, seen through the courtesy of Dr. W. E. Bruner, of Cleveland, was operated on for glaucoma by bilateral trephining in 1933. The vision continued to fail, and miotics were used. Vision was 6/200 in the right eye and 9/200 in the left. Tension was 28 in the right eye and 21 in the left (after no drops for twenty-four hours). Both disks were atrophic and cupped.

There were a few peripheric opacities of the lenses. The fields showed a large atypical cecocentral scotoma in the left eye and a superior hemianopia in the right eye. A roentgenogram revealed calcification of the internal carotid artery.

The following case does not exactly belong to this group of cases of basal sclerosis but presents a problem common to all.

CASE 11.—E. C. R., aged 60, was seen on Oct. 22, 1931. The vision in the right eye was 20/20 and the tension 22. The vision in the left eye was 20/40— and the tension 18. Both fields showed an irregular defect and a left homonymous hemianopia. A roentgenogram demonstrated an enlarged sella turcica. On December 17, an adenoma of the pituitary body was removed by Dr. Harvey Cushing. The field improved in the upper half of the left eye, and the vision was 20/20 in the right eye and 20/30 in the left.

On Nov. 1, 1934, there was some nasal contraction of the right field. The vision in the right eye was 20/20 and in the left eye 20/20. The tension was 31.

On Nov. 10, 1936, vision in the right eye was 20/20— and in the left eye 20/20. The field was unchanged. Tension in the right eye was 34 and in the left eye 28.

Dr. MacMillan, of Montreal, trephined the right eye on Jan. 18, 1937, as the field had diminished, and on May 22 the left eye was trephined and the operation was repeated on the right eye. This led to a reduction of the tension to 5 mm. Opacities of the lenses then developed, and in June the vision in the right eye was 6/15 and in the left eye 6/20.

COMMENT

The moderate rise in ocular tension in some of these cases is difficult to explain. It is not sufficient to account for the advanced change in the optic nerve, and it appears that two pathologic processes are probably present.

I have observed among patients with general arteriosclerosis several with partial atrophy of the optic nerve and marginal cupping; this suggested an arteriosclerotic process in the optic nerves. And I shall never forget the ophthalmoscopic picture of atrophy of the optic nerve with deep cupping which was observed in the days of wood alcohol poisoning when there never was a question of glaucoma. This suggested that the cupping of the optic disk without tension might be due to causes other than glaucoma.

In the presence of tension, the question of operation naturally arises. In my experience, decompression in cases of so-called basal sclerosis has been of no avail; the deterioration of field and vision proceed with the frequently added complication of cataract. This negative result of the operation can also be regarded as additional evidence that the condition is not purely glaucomatous.

There are certain cases, however, in which an operation must be done. An interesting example of this complication was observed in the case of pituitary tumor in which persistent tension subsequently developed and operation was successfully performed by Dr. MacMillan.

In my previous paper attention was drawn to sclerotic changes in certain basal vessels which were accessible to roentgenographic demonstration.

The significance of roentgen evidence of calcification of the carotid arteries has been questioned. In a series of 40 patients over 50 years of age with a variety of ocular diseases, Glees¹ found roentgenographic evidence of calcification of the carotid artery in 5 and other evidences of calcification and bone changes in 22. Calcification of the carotid artery was observed as frequently in cases of glaucoma as in cases of pseudoglaucoma or atrophy of the optic nerve.

Siebert² on roentgenographic examination of 33 patients over 60 years of age, selected without reference to their ocular disease, found changes in 50 per cent. When sclerosis of the carotid artery was present there were always arteriosclerotic changes in the retinal vessels, and patients with arteriosclerotic or hypertonic retinal changes most frequently presented calcification of the carotid artery on roentgen examination. Of 16 cases in which the roentgenogram revealed calcification of the carotid artery, true glaucoma was present in 3 and arteriosclerotic changes in the fundus in 6. On the other hand, negative roentgen findings were not unusual in cases of glaucoma without tension.

In none of the cases described in this article did the retinal vessels show unusual arteriosclerotic changes.

The significance of roentgen findings is further mitigated by the observation that compression of the optic nerve in thrombosis or calcification of the internal carotid artery does not lead to atrophy of the optic nerve.

The situation in hypophysial tumors with primary atrophy of the optic nerve is fundamentally different. There, there is enormous stretching of the optic nerve and chiasm with constriction by the crossing blood vessels, which must result in ischemia and circulatory disturbances of the nerve substance.

Best³ stated that injury to the intracranial optic nerve by arteriosclerotic changes in the carotid or ophthalmic artery in the absence of aneurysm has not been proved and drew attention to the fact that arteriosclerotic disease of the small nutrient vessels may damage the optic nerve (Henschen, Fuch and Rönne). In these cases the ophthalmoscopic picture of "glaucoma with low tension" may develop.

A similar opinion is held by Siebert² and by von Stief,⁴ who expressed the belief that arteriosclerotic nutritional disturbances of

1. Glees, M.: Arch. f. Augenh. **110**:121, 1936.

2. Siebert, P.: Arch. f. Ophth. **138**:798, 1938.

3. Best, F., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 6, p. 546.

4. von Stief, A.: Ztschr. f. Augenh. **70**:41, 1929.

the optic paths are more likely to be the cause of the atrophy of the optic nerve in cases of pseudoglaucoma than compression from calcareous changes in the basal vessels.

Marchesani⁵ suggested that the pathologic change in the optic nerve is like that in the brain (*status spongiosus*) in which gaps appear in the brain tissue when circulatory disturbances are present; in the optic nerve these changes are represented by Schnabel's cavities (*lacuniform degeneration*).

The knowledge of this question will be improved when more adequate anatomic studies of the nutritional vessels of the visual pathways are available.

CONCLUSION

Descending atrophy can be a compression product in hypophysial tumor only when the chiasm is stretched and compressed. Atheromatous carotid arteries cannot alone cause this descending atrophy, but the condition must be caused by simultaneous circulatory disturbances in the optic nerve from arteriosclerotic vascular changes.

My purpose in this paper is to emphasize again that these patients who were treated for glaucoma did not have the usual type of glaucoma in that their fields were different, the course of the disease was different, it being very slow if not stationary, and operation was of questionable benefit. The roentgenographic demonstration of a calcification of the basal vessels cannot be regarded as proof of compression of the optic nerve but suggests vascular disturbances in the nutritional vessels of the optic nerves.

5. Marchesani: *Klin. Monatsbl. f. Augenh.* 95:389, 1935.

INTERSTITIAL KERATITIS CAUSED BY SPECIFIC SENSITIVITY TO INGESTED FOODS

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AND

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The purpose of this paper is to call attention to a condition concerning which we have been unable to discover reports in the literature.

Interstitial keratitis of inflammatory type is known to be produced by syphilis, tuberculosis, Trypanosoma and the apparent invasion by a virus from without following trauma, as in deciform keratitis or ring abscess. Keratitis profunda is known to occur in debilitated persons, but the cause is obscure. There are, in addition to cases of interstitial keratitis which have been classified, a certain number of cases in which the cause is unknown. It is to a portion of the latter cases to which attention is to be called in this paper; it will be shown that interstitial keratitis of inflammatory type, with definite physical characteristics which can be recognized clinically, is caused by specific sensitivity to ingested foods.

The possibility that interstitial keratitis could be caused by a specific sensitivity to food was suggested by Tooker.¹ He said:

The effect on ocular lesions of eating protein and nonprotein foods to which a patient is sensitive apparently has not received much attention. In the two cases of tuberculous keratitis herein reported, the patients exhibited definite ocular allergic reactions following the ingestion of certain protein and nonprotein food during the course of the disease. The reactions recurred frequently enough to establish the opinion that they were not fortuitous.

Since 1930 we have seen 6 persons with interstitial keratitis who have presented a characteristic appearance and who have been relieved by removing certain articles of food from their diet. The determination of the sensitizing foods has been made by one or more of the three methods employed in the study of allergic sensitivities—by history, by elimination and by cutaneous tests. It has not been possible to employ all three methods in every case.

Read before the Section on Ophthalmology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 19, 1939.

1. Tooker, C. W.: Allergic Phenomena in Tuberculous Keratitis: Report of Two Cases, *Arch. Ophth.* **2**:540 (Nov.) 1929.

In all 6 cases at least one food can be specifically named the ingestion of which will reproduce clinical symptoms after its withdrawal from the diet has been accompanied by relief of clinical symptoms. In some of the cases more than one article of food has been found which will reproduce the symptoms. It is possible, of course, that there are still other foods which might be unknown to be, or might later become, sensitizing agents.

The disease commences with slight irritation, mild discomfort, lacrimation and redness. These symptoms continue for weeks, often with remissions, but in general they become more and more severe. They fail to respond to local treatment. After the symptoms have continued for some weeks, the vision becomes slightly hazy when the process has involved the central portion of the cornea. The vision becomes progressively more clouded, until finally it is reduced to the perception of shadows. The pain becomes more severe, until at times it is unbearable; 1 patient came at this late stage for enucleation for relief of pain.

In the early stage there is moderate ciliary injection. The slit lamp shows faint, irregular, gray or yellowish gray interstitial opacities in the cornea, at first fairly well localized in one quadrant. Small vacuoles appear early. Loops of scleral vessels soon develop deep in the corneal stroma toward the lesions. Conjunctival loops also extend into the cornea in the more superficial layers of the stroma, always beneath the epithelium however. These vessels are less numerous than in the syphilitic lesions and do not produce the "salmon patch."

As the disease progresses, the opacities increase in number and in density, extending to all portions of the cornea. Scar tissue invades the stromal layers, until the whole cornea is covered except for a fairly clear margin near the limbus, through which, however, vessels may be seen running in the corneal stroma. Dense opacities resembling calcium deposits appear late. Rarely do any of the lesions break through the anterior epithelium of the cornea to produce ulceration, although ulcers did occur in one of the late cases following overvigorous local treatment by heat, applied with a Todd cautery. No intraocular complications have been seen.

It will be seen that this condition is similar in many features to that in some of the cases of "recurrent vascular keratitis of unknown origin" reported by Doggart² in 1931. It is suggested that a search for specific food sensitivity might well have revealed an origin in some of his cases.

2. Doggart, J. H.: Recurrent Vascular Keratitis of Unknown Origin, *Proc. Roy. Soc. Med. (Sect. Ophth.)* **24**:41 (May) 1931.

The treatment for this condition is specific with the removal from the diet of all offending foods, which are ascertained by history, elimination or cutaneous test. The ciliary injection and pain disappear in forty-eight hours. Opacities which are not escharotic gradually lessen in depth, although arrest of the process is all that can be expected after actual scar tissue has appeared. The vessels in the corneal stroma continue to function for a long time, although their diameters seem to be lessened.

If the sensitizing food is taken at any time after the process is arrested and the eye is white and quiet, all symptoms will reappear within twenty-four hours, although the experimental reproduction of the disease twice is the limit of experimentation to which any patient has submitted.

The 6 cases which have provided this information are presented in the order in which they were first seen.

REPORT OF CASES

CASE 1.—Mrs. R. G., aged 49, was seen Nov. 6, 1929, with advanced interstitial keratitis. The eyes were painful, with marked ciliary injection. The vision was reduced to the detection of shadows. The disease had become progressively worse, with some remissions for eleven years.

The ulcerations occurred over areas in which deep opacities were particularly dense and followed vigorous local pasteurization. It is thought that the treatment was overzealous and caused a breaking down of the superficial epithelium. One ophthalmologist who saw the patient made a diagnosis of tuberculosis on the basis of the appearance of the corneas and a negative Wassermann reaction. We have no record of the tuberculin test at that time. The patient was given a long course of tuberculin but was not relieved. Another ophthalmologist of wide clinical experience felt that the disease was not tuberculosis and did not make a positive diagnosis. At another clinic the patient was given a series of injections of an autogenous vaccine made from intestinal material. This treatment failed to give any relief. Atropine had of course been used.

Since the patient had gone for eleven years with a diagnosis of interstitial keratitis without relief, we felt justified in making a new start in determining the causative agent. Syphilis was out of the question. The Wassermann reaction was negative, the disease developed too late in life, there was no associated intraocular complication and the eyes showed no clinical evidence of syphilis. The appearance of the corneas closely resembled the picture produced by tuberculosis; however, the patient did not have any other form of clinical tuberculosis, and an adequate course of tuberculin therapy had been given without benefit. Ulcerative keratitis could be ruled out, since ulceration had been present only three or four times in the whole eleven year course of the disease, and then only following some form of external treatment. The disturbance of the corneal layers was not so great as in deciform keratitis, the course was much longer and slower in development and there was well defined localization of the lesion.

The corneal involvement known as keratitis profunda and associated with uveitis could not be considered since there was no evidence other than ciliary injection of vessels that there was any intraocular involvement; the iris seen through the

clearer portions of the cornea was normal, for example. None of the forms of corneal dystrophies could be considered because of the definitely inflammatory character of the disease. It seemed reasonable, therefore, that some other diagnosis might be made, and that of allergic manifestation to ingested foods was tentatively adopted.

Since the equipment for adequate cutaneous tests was not readily available, the patient was sent to the hospital and her diet reduced to zero. The only local treatment was atropine and hot fomentations. At the end of two days improvement was marked, the pain having disappeared and the ciliary injection lessened. Day by day simple articles of food were added until a maintenance diet was established which did not produce ocular symptoms. The problem was outlined to a daughter, and the patient was discharged to her home. The daughter kept an accurate record of all ingested material with notes on the ocular symptoms. Various foods were added to the basic diet for variety from time to time. It was noted that pork produced an increase in the symptoms. On the basic diet the patient was free from pain and redness and the vision improved till she could care for herself. She was again seen after six years, when a photograph was taken. On one occasion an intense exacerbation of symptoms appeared while there had been no change from the basic diet. On investigation of the milk supply, the daughter found that because of food shortage the milk cattle had been given cottonseed meal. She had one cow set aside from the herd for the patient's milk supply, and the symptoms promptly disappeared.

No cutaneous tests have been made. The patient is so satisfied with her condition under the trial and error method that she has refused to undergo the tests. She has also refused an optic iridectomy, which promises her considerable improvement since there is one fairly clear area in the cornea of the right eye. The patient's only other symptom which suggests an allergic phenomenon is a "sensitive skin." The daughter suffers from severe hay fever.

CASE 2.—Mrs. B. was seen in May 1932 at the age of 75. She came in for enucleation of the right eye, since she had been told that in no other way could the intolerable pain be relieved. The entire cornea was opaque except for a narrow margin at the limbus through which scleral vessels could be seen passing into the leukoma. The eye had marked ciliary injection. The condition had been present for many years, and it had been eight years since the enucleation had been advised. The left eye showed a quadrant from 2 to 6 o'clock involved with what was obviously an earlier stage of the same process. There was moderate ciliary injection. The corneal epithelium was smooth, as it is shown clearly in the photographic high light. Deep in the corneal stroma were gray patches of varying density. The slit lamp showed these opacities to be made up partially of vacuoles and partially of diffuse white and yellowish white homogeneous deposits.

The patient had seen many ophthalmologists and had had many tests and considerable treatment, without relief; therefore she wanted the right eye removed for relief of pain.

Since all other treatment had been of no avail and since the appearance of the eyes was so similar to that in case 1, the patient was asked to submit to cutaneous tests for allergic sensitivity. She showed strongly positive reaction to wheat, strawberries and cucumbers, and a diet was prescribed excluding these items. She was seen again in three days. The pain had disappeared, and the eyes were much whiter. In a week the eyes were white. She was partly convinced that the source of her trouble was found. At the end of ten days she could not resist a strawberry shortcake, and the next day the eyes were inflamed and painful

and she was completely convinced. The exacerbation was short lived, and during the next month she was free from pain and inflammation except for two days after the ingestion of a part of an ice cream cone, which she ate before she remembered that it contained wheat. She volunteered the information that she had not known what it felt like to be really well for many years. She returned to her home city, and a letter a year later reported that her eyes were perfectly comfortable. They remained so until the patient's death in 1934.

CASE 3.—Mrs. H. D., aged 83, was seen only once, in September 1937. Her eyes had been irritated, red and painful for the past month. There was considerable conjunctival and some ciliary injection. Each cornea showed faint interstitial opacities near the limbus with loops of scleral vessels attending toward them. Several small vacuoles showed in the slit lamp beam in the stroma. Thinking that this might be an early stage of the condition seen before, we inquired into the recent changes in the patient's diet. Her doctor had discovered in the summer that she had hypertension and advised that she substitute cheese for meat. She was tested for allergic sensitivity, and a strongly positive reaction appeared with cheese. Her eosinophil count in the blood was 4 per cent. She was requested to eliminate cheese from her diet and to return in a week for inspection. She notified us that her eyes were well, but she has never returned.

CASE 4.—M. R., aged 18, seen on Dec. 11, 1937, complained of blurred vision and pain in the left eye of three years' duration. She had been given a pair of glasses, which had not helped. She had also been advised that the soreness was caused by an infected antrum. This was drained, but no improvement in the ocular condition followed. The left eye showed moderately dense interstitial opacities extending into the pupillary area. There was considerable formation of vacuoles and loops of new vessels deep in the stroma. Slit lamp inspection showed a small peripheral opacity in the other eye, to which ran a new vessel.

The patient was referred for physical examination, which did not reveal any clinical abnormality. Cutaneous tests showed a positive reaction to pork and a strongly positive reaction to egg white. She was given the Rowe elimination diet, group 1, with articles of group 2 to be added one at a time as long as the eye remained white. By Jan. 8, 1938, her eyes were white. When she was seen on January 29 her eyes were white. On this date she had a slight exacerbation following the drinking of a glass of coca-cola. In the summer she moved to another locality and has not been seen since. We do not feel that we have a complete list of the foods to which she may be sensitive.

CASE 5.—Miss F. W., aged 33, was seen on July 29, 1938, complaining of an irritated red eye. The eye showed interstitial infiltration of the cornea in the lower nasal quadrant, with the formation of vacuoles and new vessels. There was rather marked ciliary injection. She said that she had a similar attack some years before, which had persisted a long time. At the time of the previous attack a physical examination gave negative results, and the Wassermann test was negative. She was given the Rowe diet, group 1, articles from group 2 to be added gradually. She returned to her position as school teacher in another city and has reported by letter that her eyes have been well except for one occasion, when she broke her diet by eating chocolate candy. This was followed by a return of her symptoms. She also feels that wheat foods definitely increased the symptoms. Cutaneous tests will be carried out at the first opportunity.

CASE 6.—Mrs. C. H. M., aged 42, was first seen on Dec. 3, 1938, complaining that the left eye had been red for the past ten days. There was only slight soreness, and she objected only to the appearance. The sector of the cornea at 8 o'clock showed a faint interstitial haze with four somewhat more dense patches. Coming to them from the sclera were several loops of new deep blood vessels. Inquiry was made into changes of diet of recent date, and it was discovered that three weeks before rye biscuits had been substituted for bread to aid reduction in the weight. We asked her to eliminate rye from her diet and return for inspection. In the discussion it was revealed that the patient's daughter had been relieved of allergic rhinitis by eliminating milk from the diet. On December 6 the eye was white. The patient was referred for a scratch test for rye. It was mildly positive. The nasal secretion showed 8 per cent eosinophils. The patient was asked on December 10 to resume the eating of the rye biscuits. On December 12 she reported on the phone that her eye was again red and sore and that she wanted to discontinue the experiment. The eye was again white on December 17. On December 21 she had an intradermal test for rye. This produced a violent reaction, not only locally but focally, both eyes becoming very red. The eye had quieted by December 27, showing only mild ciliary injection. The patient announced that she had experimented enough with rye.

It may easily be seen that rather than keep these patients on elimination diets more work must be done in some instances in order to establish the specific foods to which the patient is sensitive. However, there is no question that interstitial keratitis can be produced by specific sensitivity to ingested foods and that the condition may be relieved immediately clinically by elimination of these foods. Naturally, the earlier the diagnosis is made the better will be the result, since actual proliferation of scar tissue into the corneal stroma becomes a permanent opacity.

We urge that patients with interstitial keratitis of obscure cause be studied for specific sensitivity to foods. We naturally hope that our observations will be verified.

ABSTRACT OF DISCUSSION

DR. ALBERT D. RUEDEMANN, Cleveland: To the group of conditions that will produce interstitial keratitis might be added undulant fever, to which attention was called recently by Dr. John Green, of St. Louis.

Undulant fever simulates tuberculosis in all portions of the eye. I had the experience of going over a series of 137 cases of undulant fever. There were ocular manifestations in 7 of them.

In regard to interstitial keratitis in association with ingested food, one must be careful to make the diagnosis by the elimination of all other possible sources of infection and the solution of all other possible causes of interstitial keratitis.

My experience with ocular manifestations of allergy does not bear out the fact that interstitial keratitis or other manifestations are observed in elderly persons. It usually occurs in the early decades of life and not later; corneal involvement that is seen in persons who are in the

late decades is usually due to some senile dystrophy or food deficiency. I think that interstitial keratitis must be considered a deficiency disease as well as ocular allergy.

Another factor must be brought to mind and that is that the diagnosis of ocular allergy is made by exclusion, by a careful history, by exacting cutaneous tests and, lastly, by elimination diet. I have been unfortunate in not being able to rule out ocular manifestations of allergy by elimination diet in many of the cases in which I thought this condition existed. History is a better factor, because one obtains the history of other allergic states, associated rhinitis or associated gastrointestinal disturbance. Food is the most difficult factor to rule out. The history of the ingestion of certain foods is not always a criterion to go on.

The repeated ingestion of small amounts of a food to which the patient is sensitive may be the causative factor, even though his reaction to that food is slight, whereas the infrequent ingestion of large amounts of food does not tend to produce many ocular changes because the periods between the times of ingestion are too long and the eye tends to clear up.

It is repeated chronic attacks that produce the vascularization. All patients with severe superficial keratitis who have desquamation and deep injection of the ocular conjunctiva will show some vascular loops extending into the cornea if the eyes are studied with the slit lamp. If these attacks persist, some interstitial vascularity will occur in those patients with contact dermatitis which is not due to deep inflammation or to the general condition. However, as one goes deeper into the eye it will be found that certain foods will play a definite part in the production of the ocular manifestations of allergy.

Foods, primarily wheat, butter and foods that are taken repeatedly and not infrequently, are usually the causative agents.

Elimination of these foods from the diet does not always eliminate the ocular condition for the simple reason that most of the patients with ocular allergy have secondary allergic states which in themselves may cause the process in the eye.

I must draw attention to the fact that if a diagnosis of ocular allergy is to be verified the patient must be studied carefully. Every possible angle should be considered, otherwise ophthalmologists will fall into the same group or the same class with the otolaryngologists, who studied allergy over a period of years, giving injections for every form of change in the nose.

DR. ABBOTT M. DEAN, Council Bluffs, Ia.: I agree with Dr. Ruedemann heartily that it is difficult to establish the ingestion of food as a cause of allergy. Careful study is required, as he has pointed out. It so happens that in these particular cases I was satisfied with the results because the patients did get well. The ocular manifestations were probably not caused by tuberculosis or syphilis, since treatment was not given for any other condition.

In considering the question of age, it is true that allergy is usually discovered in early life, but in case 6, for example, the patient had never eaten rye before. Therefore, she had not had an opportunity to find out whether she was sensitive to rye or not.

A NEW METHOD OF APPLYING RADON SEEDS FOR OCULAR DISORDERS

JOSEPH WALDMAN, M.D.

PHILADELPHIA

Radium treatment of retinoblastoma had been carried out by Axenfeld¹ in 1914, Uhthoff² in 1917, Schoenberg³ in 1919, Knapp⁴ in 1920 and others⁵ in succeeding years. But real impetus to this form of treatment was given by Moore, Stallard and Milner⁶ in 1931. At that time they described a technic by which radon seeds were inserted directly into the growth and also sewn to the overlying sclera. In 1932 Stallard⁷ published a truly monumental work on the use of radiant energy in the treatment of ophthalmic disorders. He described in detail methods of direct insertion of radon seeds and suturing to the overlying sclera. McDowall and Marks⁸ in 1932 reported a case in which radium needles were inserted through incisions in the upper lid into the orbit near the base of the tumor. In 1934 Barkan⁹ described a method of applying radon seeds to the overlying sclera by embedding them in dental compound.

Moore¹⁰ in 1935 described a method of applying radon seeds to the overlying sclera by means of a collar of sharp spring spikes around the seed. These served to fasten the seed to the sclera.

From the Department of Ophthalmology, Service 2, Jefferson Medical College Hospital.

1. Axenfeld, T.: *Klin. Monatsbl. f. Augenh.* **17**:426, 1914.

2. Uhthoff, W.: *Deutsche med. Wchnschr.* **43**:1023, 1917; *Klin. Monatsbl. f. Augenh.* **49**:241, 1920.

3. Schoenberg, M. J.: *Arch. Ophth.* **48**:485, 1919.

4. Knapp, A.: *Arch. Ophth.* **49**:575, 1920.

5. (a) Kusama: *Am. J. Ophth.* **3**:244, 1920. (b) Janeway, H. H.: *Arch. Ophth.* **49**:156, 1920. (c) Weeks: *ibid.* **49**:241, 1920. (d) Chase, S. B.: *Am. J. Ophth.* **3**:806, 1920. (e) Poyales and Pajares: *Arch. d'opht.* **38**:122, 1921. (f) Kumer, L., and Sallman, L.: *Die Radiumbehandlung in der Augenheilkunde*, Berlin, Julius Springer, 1929. (g) New, G. B., and Benedict, W. L.: *Am. J. Ophth.* **3**:224, 1920.

6. Moore, R. F.; Stallard, H. B., and Milner, J. G.: *Brit. J. Ophth.* **15**:673, 1931

7. Stallard, H. B.: *Radiant Energy as Agent in Ophthalmic Disorders*, London, George Pulman & Sons, Ltd., 1932.

8. McDowall, V., and Marks, E. O.: *Brit. J. Ophth.* **16**:686, 1932.

9. Barkan, H.: *Bilateral Glioma Tested by Radium*, *Arch. Ophth.* **11**:20 (Jan.) 1934.

10. Moore, R. F.: *Tr. Ophth. Soc. U. Kingdom* **55**:3, 1935.

In 1936 Stallard¹¹ published further observations on the suturing of radon seeds to the globe.

In 1938 Stallard¹² described his latest method, which consisted of embedding the seed in a piece of Stent's dental wax 4 mm. wide and 2 mm. thick. This was then molded to the sclera and sutured in place overlying the growth.

The experience of the average ophthalmic surgeon will bear out, I believe, the fact that suturing in the orbit, especially on the posterior half of the eyeball, is rather a difficult procedure technically. This is true especially when the region surrounding the optic nerve is the field of operation.

In view of the difficulties encountered, a new method of applying radon seeds to the overlying sclera is suggested.

Naturally, an accurate localization of the growth is of paramount importance. The operator has a wide field of choice in this matter. Frequently, owing to the fact that cooperation of the patient cannot be obtained because of his age, localization will have to be done with the patient under a general anesthetic.

The amount and number of radon seeds to be used are important and should depend on the size and location of the growth. The tumor mass may extend into the vitreous. This distance should be calculated and borne in mind when the seed is applied. Barkan used two seeds of 5 millicuries each. Stallard has used one to four seeds of 2 millicuries each.

It is not the purpose of this paper to discuss the dose. This is left to the discretion of the surgeon.

TECHNIC OF APPLICATION

After the proper aseptic and antiseptic preparation, the conjunctiva is incised 5 mm. from the limbus. The portion attached to the limbus is undermined. Then Tenon's capsule is opened, and the area to be treated is exposed and freed from the overlying capsule. Deep retractors are of considerable help in this step.

With the proper exposure made, the applicator with the radon is applied.

The applicator is made of rolled sheet "fine" silver or platinum 0.5 mm. in thickness, with rhodium or chromium finish. This metal is suggested because it can easily be bent to conform to the curvature of the eyeball and can be sterilized by boiling. One-half millimeter thickness will cut out the beta rays and perhaps a negligible amount of gamma radiation. It is not affected by reaction with body fluids or tissue. The

11. Stallard, H. B.: *Brit. M. J.* **2**:962, 1936.

12. Stallard, H. B.: *Brit. J. Ophth.* **22**:604, 1938.

edges should be smooth and free of sharp points. The width of the band suggested is not more than 5 mm. The length depends on the site of growth. The end used as the anchor is perforated on either side of the band in parallel rows to permit passage of suture material and needle. At least two sets of perforations 3 mm. apart are suggested to insure good fixation and to prevent moving after the applicator is applied. Black silk is recommended because it is easily seen and will facilitate removal.

The actual receptacle for the radon seed is on the distal end of applicator. It is merely a depression made by means of a rectangular punch, the dimensions of which are such as to receive the seed snugly to prevent shifting.

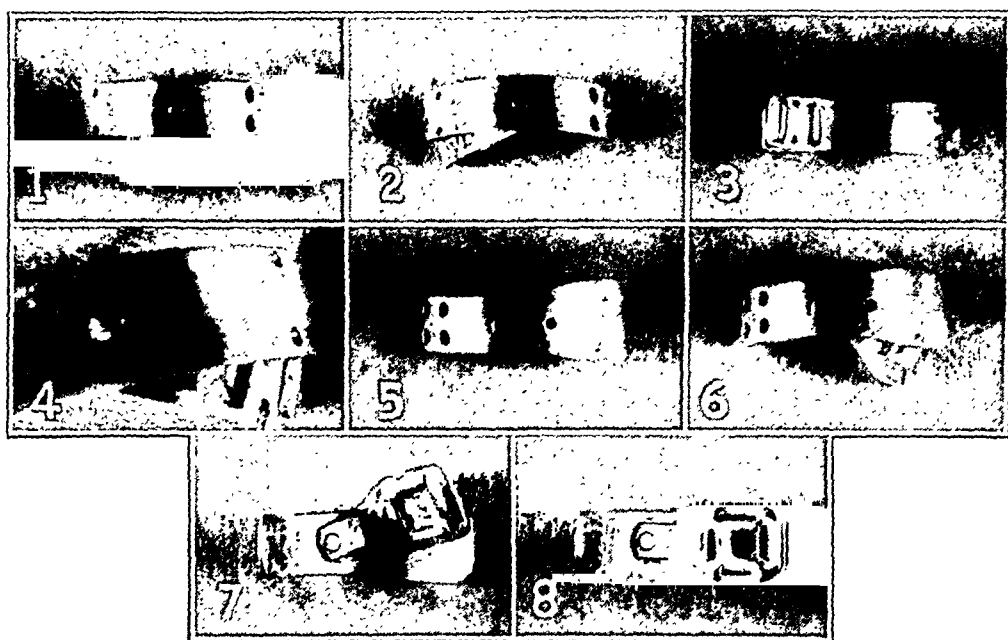


Fig. 1.—The applicator for two seeds is shown in 1 to 4 (inclusive), the applicator in 4 being enlarged, and the applicator for 4 seeds is shown in 5 to 8.

This is covered by another thin sheet of silver or platinum which extends from one half the length of the applicator to the distal end on the convex side. This will insure a smoother area of contact with the sclera. (Figure 1, 2, however, shows the shorter arm on the concave side.) It is fastened to the longer arm of the applicator by a fine silver rivet, hammered on both sides to prevent slipping through the holes made to receive it. This will permit a sliding or scissors movement of the short arm over the long arm.

The spring effect can be increased by merely increasing the bend of the larger arm slightly. This will insure the safe closure of the receptacle containing the seed.

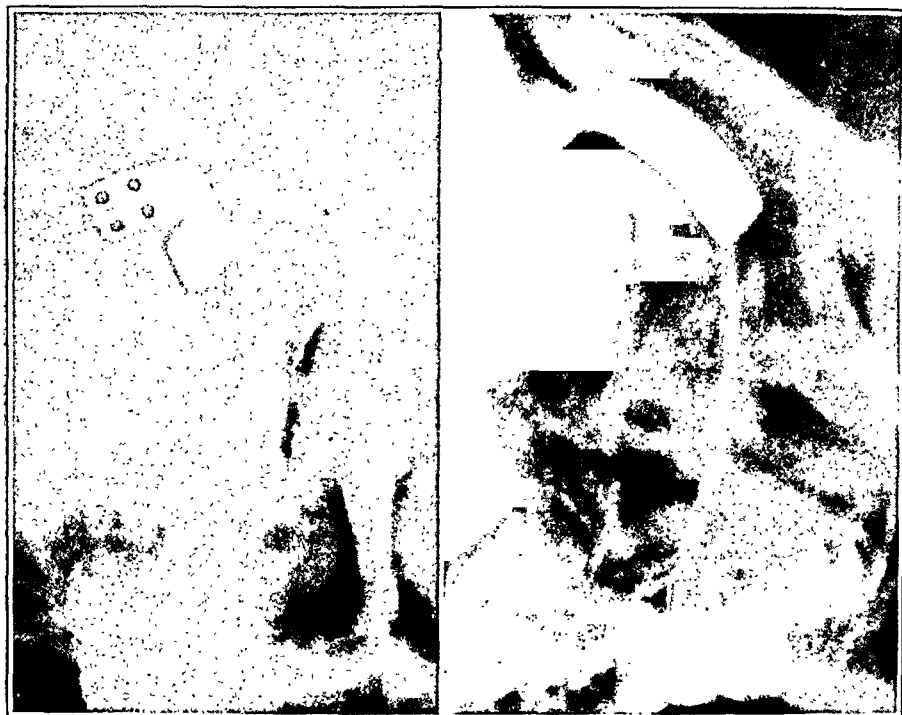


Fig. 2.—Anteroposterior and lateral roentgenogram (slightly enlarged) showing the applicator in position.

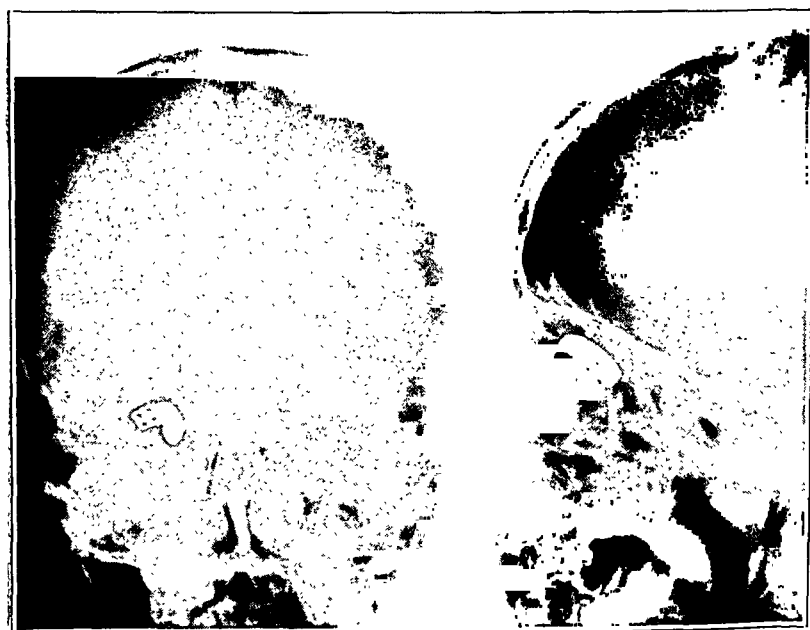


Fig. 3.—Anteroposterior and lateral roentgenograms (reduced) showing applicator in position.

To be doubly certain that the radon seed does not come out, tying the two arms through the perforation at the distal end securely with silk is recommended. To prevent secondary radiation to the surrounding tissues, some plastic adhesive sheet rubber (sterilized by boiling) is molded over both ends.

It is suggested that several such applicators of different lengths be at hand at the time of operation. The longest, conforming to the curvature of the eyeball, should extend from the limbus to a point close to the optic nerve. The shorter ones can be "lengthened" in effect by merely moving them the required number of millimeters from the limbus in the direction of the growth.

If more than one seed is necessary, then the applicator can be made multicellular, depending on the number of seeds used. Or if a barrage is desired, a circular, square, triangular or T-shaped distal segment can be used.

Any competent jeweler can make these applicators with little difficulty.

It is believed that the applicator described here will eliminate difficult technical suturing in the posterior half of the eyeball.

After being placed in the proper position, it is sewn to the sclera near the limbus. Tenon's capsule and conjunctiva are closed. The applicator is thus "buried." After the conjunctival sutures are removed, and the conjunctiva is reopened at the end of the period of application, the sutures are removed from the proximal end of the applicator and the conjunctiva is closed again.

Roentgenograms are suggested with the applicator in situ to be sure that the seed has not fallen out. The latter possibility is remote if the precautions mentioned are observed.

Dr. Charles E. G. Shannon, of Philadelphia, has permitted me to assist him in using this method for one of his patients. This will be reported at a later date.

SUMMARY

A simple safe method of applying radon seeds to the sclera overlying an intraocular growth is described.

It may be used in cases of bilateral involvement as a preoperative measure or in the remaining eye with any intraocular disturbance in which it is thought that radium therapy is indicated.

CERTAIN RETINOPATHIES DUE TO CHANGES IN THE LAMINA VITREA

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AND

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Hyaline bodies on the lamina vitrea of the choroid, the *Drusen* of the Germans, are familiar to every one who uses the ophthalmoscope or has studied ocular histopathology.

Their histogenesis has been a matter of discussion, and the subject has recently been brought up to date by Rones.¹ It seems not to be universally known, however, that closely packed aggregations of hyaline bodies in the macular region may in some middle-aged or elderly persons result in more or less serious disturbance of vision. It is to this distinct clinical picture that we wish to call attention. Some notes will be added on two other conditions in which degenerative changes of the lamina vitrea are presumably one or perhaps the only causative factor.

Hyaline bodies occur frequently in otherwise normal eyes, becoming larger and more numerous as age progresses but being found occasionally in young persons. They become larger and more numerous in eyes which have undergone injury or chronic inflammation, especially in phthisic eyes, in which they are usually recognized only in sections. Rones expressed the belief that a distinction should be made between the senile variety and that seen in diseased eyes. For the former he seems content to accept the explanation of Coats² that they are deposited by the pigment epithelium in a process similar to that responsible for the deposition of the cuticular layer of the lamina vitrea, which may be considered as a specialized basement membrane of the pigment epithelium. The variety seen in diseased eyes, he stated, arises by hyaline degeneration of the pigment epithelial cells themselves. Verhoeff³ disagreed with him and expressed the belief that both types

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Read before the Seventy-Fifth Annual Meeting of the American Ophthalmological Society at Hot Springs, Va., June 7, 1939.

1. Rones, B.: Formation of Drusen of the Lamina Vitrea, Arch. Ophth. **18**:388 (Sept.) 1937.

2. Coats, G.: Roy. London Ophth. Hosp. Rep. **16**:164, 1904-1905.

3. Verhoeff, F. H., in discussion on Rones.¹

are best explained by the deposition theory of Coats, which Parsons⁴ and most other modern students of the subject are inclined to accept.

Rones recognized that "when the dots are numerous and fused the condition can result in diminution of vision by pressure on the rods and cones." He did not enlarge on this statement, however, and little attention has been given of late, at least in the literature, to this cause of visual disability. In England it was first described in 1876 by Jonathan Hutchinson,⁵ who with Tay reported 3 cases in sisters and at least 4 other isolated cases of a similar nature. Hutchinson recognized that the white bodies were probably allied to the "colloid" bodies of the lamina vitrea and that they must damage the retina by pressure. In England the condition was known as Tay's senile guttate choroiditis and later also as Doyme's honeycomb choroiditis, in honor of Doyme,⁶ who in 1899 reported 4 cases seen in a family of 8. He later⁷ saw the other affected families, and his contributions familiarized English ophthalmologists with the picture and allowed it to be distinguished from other conditions grouped with it by Hutchinson. Doyme considered the white deposits to be a form of exudation into the choroid and not an inflammatory condition. When Treacher Collins⁸ was able to examine sections from the eye of one of Doyme's patients the true nature of the white spots was revealed as due to the deposit of a "hyaline substance between the retina and choroid." In this case, that of a man who died at the age of 80 with vision reduced to the counting of fingers, the hyaline deposit was described as commencing on each side of the disk close to its margin and extending inward from it 2 disk breadths. Its inner or retinal surface presented several rounded nodular elevations with depositions between them. Over these nodules the pigment epithelium was absent and the retinal tissue was much disorganized. The rod and cone layer was entirely destroyed, as was much of the outer nuclear layer. Collins considered the hyaline substance as of similar origin to that seen in so-called drusen, while the retinal changes were explained as due to pressure of the hyaline substance. Collins⁹ later classified the familial form of this condition among the hereditary abiotrophies.

Most of Doyme's patients were seen at the age of 40 or more, but 1 girl of 20 showed the condition. Vision was little affected in the early stages, but central vision was gradually reduced, being limited to the counting of fingers in some persons who lived to be 80 or more.

4. Parsons, J. H.: *The Pathology of the Eye*, London, Hodder & Stoughton, 1905, vol. 2, p. 470.

5. Hutchinson, J.: *Roy. London Ophth. Hosp. Rep.* **8**:23, 1875.

6. Doyme, R. W.: *Tr. Ophth. Soc. U. Kingdom* **19**:71, 1899.

7. Doyme, R. W.: *Tr. Ophth. Soc. U. Kingdom* **30**:93 and 274, 1910.

8. Collins, E. T.: *Ophthalmoscope* **11**:537, 1913.

9. Collins, E. T.: *Internat. Cong. Ophth.* **1**:103, 1922.

Similarly affected families were reported by Butler,¹⁰ Mould¹¹ and Holthouse and Batten.¹² Tree¹³ recently collected data on 25 cases from the records of the Oxford Eye Hospital, including Doyne's cases and a number of new ones. Twenty of the patients were members of 5 affected families, while in 5 cases no family history was obtained. From his pictures and descriptions, the condition would seem to be the same as that in which we are interested, except that the development of the hyaline bodies reached a more extreme degree than was seen in any of our cases except case 6, and as a result vision was often poorer than in our cases.

As English investigators were interested especially in the hereditary nature of the condition, few isolated nonfamilial cases were reported by the English authors, whereas in most of the other cases found in the literature a hereditary factor has not been noted.

On looking outside the English literature, one finds, as with so many other retinal conditions, the best review of early cases in Leber's monograph,¹⁴ and some of these are of a considerably earlier date than those in the English reports. Leber recognized that extensive drusen in some cases cause visual disturbance. He reported a personal case in a woman of 84 whose vision was reduced to the counting of fingers. He referred to Masselon's case, not otherwise accessible, in a man of 82 with vision of 1/3. Müller,¹⁵ in the histologic study of drusen which he reported in 1855, expressed the view that bodies of this size must in some cases cause damage to the retinal function. The first clinical case to be reported, however, in which such damage was observed was that of Liebreich¹⁶ in 1858. His patient was a man of 26. Next was the case of Nagel,¹⁷ reported in 1868, with the first fundus picture which we could find demonstrating the condition. In 1875 he obtained sections of the eyes of a patient who had died at the age of 73. These proved that the dots seen in life were hyaline deposits on the lamina vitrea, which had destroyed the pigment epithelium over them and must have been responsible for the bilateral amblyopia. The shiny crystals seen in life were shown to be deposits of calcium carbonate embedded in the hyaline bodies. Caspar¹⁸ in 1892 described the case of a man of 68

10. Butler: *Tr. Ophth. Soc. U. Kingdom* **30**:94, 1910.

11. Mould, G. T.: *Tr. Ophth. Soc. U. Kingdom* **30**:189, 1910.

12. Holthouse and Batten: *Tr. Ophth. Soc. U. Kingdom* **17**:62, 1897.

13. Tree, M.: *Brit. J. Ophth.* **21**:65, 1937.

14. Leber, in von Graefe, A., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 17, chap. 10 A, p. 2017.

15. Müller, H.: *Arch. f. Ophth.* (pt. 2) **2**:1, 1855.

16. Liebreich, R.: *Arch. f. Ophth.* **4**:286, 1858.

17. Nagel, A.: *Klin. Monatsbl. f. Augenh.* **6**:419, 1868.

18. Caspar, L.: *Klin. Monatsbl. f. Augenh.* **30**:284, 1892.

with vision of 6/18 in one eye and opacities of the lens in the other eye which reduced the vision to perception of hand movements.

Nuel¹⁹ in 1908 reported a case, and in 1912 he was able to examine sections of the eyes of the patient and to mention 4 other personal cases. He called the condition "degenerescence pommelée de la macula," and both the clinical and the histologic picture coincided in all essentials with those described by Collins. He pointed out that metamorphosia is an early symptom, followed by central scotoma and amblyopia, which becomes stationary after a time, leaving peripheral vision unimpaired. In his sections he found the pigment epithelium raised by a thin membrane of clear, solid homogeneous substance occupying the entire macular area and showing rounded elevations. The pigment epithelium over these was less altered than in Collins' case, being only slightly thinned over the elevations, and the choroid was normal. The cones in the fovea were elongated and degenerated. While recognizing a similarity between the homogeneous deposit and that of drusen, he pointed out certain differences. The most important is that drusen are sharply elevated and isolated from each other, while in his case the elevations sloped gradually and were connected by a continuous membrane of similar material resting on the lamina vitrea, the same picture seen by Collins. From his description of the rosy pink color of the spots seen in the fundus, it is evident that the deposits were much less prominent than those in Collins' and in other cases, which were white and often shiny, having pushed through the pigment epithelium and into the retina. Another report of a histologic examination was made by Silva.²⁰ The patient had been seen a few days before death by Fuchs, and the characteristic picture was described, although no complaint of poor vision had been made. The pigment epithelium was intact over the deposits in this case and the retina was not damaged except for some questionable changes in the layer of rods and cones.

In Bedell's atlas²¹ four photographs of this condition are shown. One of the affected patients was 51 years of age and the other 3 were all 73 years of age. All showed a moderate decrease in vision, and the condition of 3 was classified as Tay's choroiditis, although no mention of heredity is made in the brief notes.

In going through our records for the past ten years, during which time special note was made of this condition in the diagnosis files, 16 cases were found which undoubtedly fit the picture which may be called "central retinopathy due to hyaline deposits." It is our impression that a number of others have been seen but have escaped proper tabulation. We have no records of the frequency with which hyaline bodies alone

19. Nuel, J. T.: Arch. Ophth. 28:737, 1908.

20. Silva, R.: Klin. Monatsbl. f. Augenh. 49:378, 1911.

21. Bedell, A. J.: Photographs of the Fundus Oculi, Philadelphia, F. A. Davis Company, 1929, plates 20-21.

are found, but their incidence is, of course, many times greater, so much so that they may be considered one of the frequent ocular signs of old age, occurring in certain persons, like other senile stigmas, at the age of 40 or even less. These cases are summarized in table 1.

TABLE 1.—*Data on Cases of Retinopathy Due to Hyaline Deposits*

Case	Sex	Age	Vision with Best Correction	Ophthalmoscopic Findings
1	F	60	R. E., 20/25 L. E., 20/30	Peripheral lenticular opacities; dense hyaline deposits in both maculas
2	F	59	R. E., 20/20 L. E., 20/15	More pigment displacement and metamorphopsia in left eye; hyaline deposits denser in left eye
3	F	49	R. E., 20/15 L. E., 20/25	R. E., some hyaline deposits; L. E., more hyaline bodies in macula with pigment displacement
4	F	80	R. E., 20/30—1 L. E., perception of hand movements	Mature cataract in left eye; few peripheral opacities of lens in right eye with dense hyaline deposits; unusual pigment deposits on cornea of each eye
5	F	69	R. E., 20/25 L. E., 20/25	Few opacities of vitreous; dense small hyaline deposits in both maculas
6	M	74	R. E., 20/200 L. E., 10/200	Some peripheral lenticular opacities; dense hyaline deposits in both maculas
7	F	74	R. E., 20/25 L. E., 20/30—	Retinal arteriosclerosis
8	F	70	R. E., 8/70 L. E., 20/25—2	Nuclear cataract in right eye; hyaline deposits in left eye
9	F	71	R. E., 20/30 L. E., 20/20	Dense hyaline bodies in right macular region; central scotoma in right eye only
10	F	67	R. E., 20/30 L. E., 20/25	Large hyaline deposits in macular areas; red area in right fovea developed in four years; vision reduced to 20/200
11	F	65	R. E., 20/30 L. E., 20/30	Dense hyaline deposits and fine pigment displacement in both macular regions
12	M	33	R. E., 20/20 L. E., 20/20	Dense small hyaline deposits in both central areas; slight pigment displacement
13	F	60	R. E., 20/50 L. E., perception of light	R. E., dense hyaline deposits and fine pigment changes in macula; L. E., thrombosis of central vein
14	F	72	R. E., 20/30 L. E., 20/40	Diffuse fine hyaline deposits in both maculas; no pigment changes
15	F	48	R. E., 20/13 L. E., 20/15	Dense hyaline deposits and fine pigment displacement in both maculas
16	F	56	R. E., 20/15 L. E., 20/15	Many hyaline deposits in both macular areas; slight pigment disturbance
17	F	55	R. E., 20/100 L. E., 20/40	R. E., nuclear cataract prevents view of fundus; L. E., some nuclear sclerosis; dense hyaline deposits and moderate pigment changes in macula
18	M	45	R. E., 20/70 L. E., 20/100	Dense hyaline deposits in both maculas

It is seen that some cases are included in which there was slight visual loss. The patient in case 2, for example, had vision of 20/20 in the right eye and 20/15 in the left. She was conscious, however, that vision had been less distinct in the left eye for the past year, and a small relative central scotoma could be made out in the left field only. Hyaline deposits in both macular areas had been seen at examinations two and three years before, being especially dense in the macular regions. In

1939 for the first time some pigment displacement could be seen about the deposits. This was slight in the right eye but more marked in the left.

Cases 1, 2, 3, 4 and 5 illustrate the early stages of the condition, all patients being certain of a definite loss of vision, usually with metamorphopsia. In all cases the hyaline deposits were especially dense in the region surrounding the nerve and including the macula. Deposits of pigment were finely granular and often slight but were present in all. The peripheral fields were normal, changes being limited strictly to the central area. The hyaline deposits were always deep to the retinal vessels and varied in size from large ones three to four times the diameter of the large retinal arteries to those less than the diameter of an artery. Only those cases were included in which the deposits were unusually numerous and close together and involved the macular area itself. A number were excluded because of central opacities of the

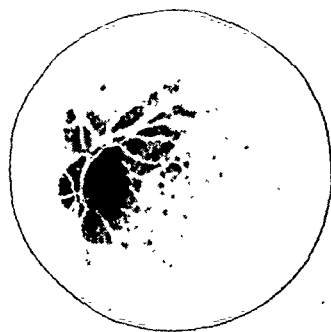


Fig. 1 (case 12).—Retinopathy in the right eye due to hyaline deposits.

lens which could account for the visual defect. A few in which there were opacities of the lens were included, but only those in which the fundus of at least one eye could be seen with perfect clearness and in which the opacities of the lens could not account for the visual defect present. General findings in these cases showed nothing of importance. Arteriosclerosis and hypertension were present no more frequently than could be expected in this age group, all of the patients except 1 being over 48 years of age, the average age being 65. One patient (case 12) was a man of 33 (fig. 1).

There was a tendency for vision to be more affected in the older patients, but this was by no means constant. Both eyes showed hyaline deposits in all cases, but in some the condition had progressed to the stage of retinal degeneration in only one eye.

In case 10 vision dropped from 20/30 in the right eye and 20/25 in the left eye at the age of 67 to 20/200 in the right eye and 20/25 in the left eye four years later. A small red area could be seen in the right fovea, where thinning of the retina was most extensive. In case 17

at the age of 55 vision was reduced to 20/40 in the left eye chiefly as a result of the deposits (fig. 2). In case 18 at the age of 45 vision was 20/70 in the right eye and 20/100 in the left eye with no other cause apparent except the dense hyaline deposits. Hemorrhages were never seen in the uncomplicated cases. In case 13 vision was 20/50 in the right eye at the age of 60. The left eye had suffered thrombosis of the central vein. The greatest reduction of vision due to the hyaline deposits alone was in case 6, that of a man of 74. Here a few peripheral opacities of the lens were also present, but they did not interfere with a clear view of the fundus. Vision was 20/200 in the right eye and 10/200 in the left eye.

No history of a hereditary factor was obtained in any of our cases. Other members of the family were not usually examined, however, and hence the possibility of such a factor cannot be excluded. Progress of the visual loss was exceedingly slow in the patients observed for a number of years.

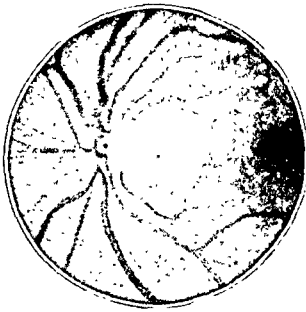


Fig. 2 (case 9).—Retinopathy in the right eye due to hyaline deposits.

In differential diagnosis the only two conditions which need be considered are: senile cystic macular degeneration and so-called Gunn's dots. While the former condition may be associated with a few hyaline deposits in the neighborhood of the macula, we believe that it has not been described in eyes showing the large number of deposits just in the macular region illustrated in our cases. In cystic degeneration the loss of vision is usually much more marked than would be expected from the fundus picture, while in degeneration due to hyaline deposits the reverse is the case. In case 9, for example, the deposits were exceedingly extensive and closely packed, with rather marked pigment displacement, yet vision was 20/30 in the poorer eye and it had shown little loss during two years' observation²² (fig. 3). It is easy to explain

22. Since writing these facts the patient has returned with vision reduced to 4/200 in the right eye. She stated that vision failed six months before. A mass of newly formed tissue is present in the macular area with hemorrhages in the deeper layers. This places the case in the class described in table 2, in which hyaline deposits were associated with central disk-shaped retinopathy.

this when it is remembered that degeneration due to hyaline deposits is purely the result of pressure, while in cystic degeneration the retinal elements themselves are the seat of a primary degeneration. The great difference in prognosis in the two conditions seems to us of no little importance, since in these two conditions an accurate prognosis is about all which can be offered to the patient. In degeneration due to hyaline deposits a slow progress undoubtedly does occur which may at an advanced age make reading impossible, but this progress is much slower and the degree of visual loss usually much less severe than in cystic degeneration. With the Friedenwald ophthalmoscope or with red-free light the finer diagnostic changes in the inner retinal layers can be seen in cystic degeneration.

As regards Gunn's dots, there should really be no difficulty, but statements seen in the literature suggest that the matter is not universally clear. Thus Horniker and Bailliart both referred to Gunn's dots and drusen as if they were synonymous. We can do no better than to quote



Fig. 3 (case 17).—Retinopathy in the left eye due to hyaline deposits.

Gunn's original description.²³ They are, according to his article, "very discrete, yellowish white, shining dots in the retina for some distance around the disk. These dots are remarkably equidistant from one another and are situated anterior to the largest retinal blood-vessels, each being less than $\frac{1}{5}$ the diameter of a large vessel." They were seen in young persons as well as in older ones in his cases; they showed no tendency to progress and did not affect vision. The histologic picture of Gunn's dots has not, to our knowledge, been studied, but their location is definitely in the innermost retinal layers or even in the internal limiting membrane itself. This should suffice to distinguish Gunn's dots from the hyaline bodies of the lamina vitrea, which are always deep to the retinal vessels and usually much larger.

A third condition may be mentioned, retinitis albescens punctata. In this condition numerous shiny white deposits are scattered all over the fundus, being located usually in the inner retinal layers. The typical

23. Gunn, D.: Tr. Ophth. Soc. U. Kingdom 3:110, 1883.

condition is associated with night blindness with contraction of the visual fields, so it apparently represents a primary degeneration of the visual cells resembling retinitis pigmentosa, except that pigment deposits are scanty or absent.

In addition to these typical cases, in most of which there was slight or only moderate visual loss, we found records of 9 patients in whom the occurrence of unusually dense hyaline deposits in both eyes was associated with the central disk-shaped retinopathy of Kuhnt and Junius in one eye or in both eyes. As will be seen in table 2, in cases 10 and 14 there were raised connective tissue masses in both macular regions, while in the others such masses were present in only one eye, the other eye showing only a rather marked reduction of vision associated with extensive hyaline bodies. In case 15 there were hemorrhages in both macular regions, but with the formation of new connective tissue only in the right eye (fig. 4). In the left eye vision has dropped only from

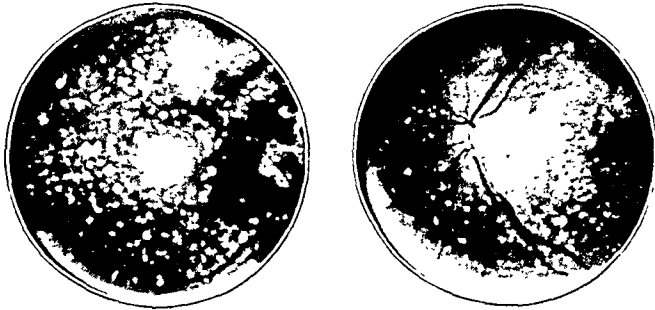


Fig. 4 (case 21).—Central disk-shaped retinopathy due to hyaline deposits.

20/30 to 20/40 during two years' observation. The patient showed the most extensive development of hyaline bodies that we have seen. They were large, and many were brilliantly refractile. He had been treated for syphilis, had moderate hypertension and had had occasional periods of increased intraocular tension. Hemorrhages were seen in only one other case, case 14. They were seen in none of the cases cited in table 1.

During the time in which these cases were seen, 35 cases of typical central disk-shaped retinopathy have been seen without the presence of hyaline bodies. In most of the cases both with and without hyaline deposits evidences of generalized hypertension and arteriosclerosis have been no more marked than is customary in the age group affected. The retinal vessels themselves have shown, as a rule, surprisingly slight evidences of arteriosclerosis, the arteries being abnormally small in few of the series. A number showed moderate arteriovenous compression, but none showed hemorrhages or deposits outside of the central area. In several the well known association with circinate retinopathy was noted.

Since it is generally agreed that an infectious origin can be ruled out in the cases of central disk-shaped retinopathy, it has naturally, along with other fundus changes occurring in old age, been ascribed by many observers to arteriosclerosis of the retinal or choroidal vessels. Kuhnt and Junius,²⁴ although they recognized the absence of marked

TABLE 2.—*Data on Cases of Central Disk-Shaped Retinopathy Associated with Extensive Hyaline Deposits*

Case	Sex	Age	Vision with Best Correction	Ophthalmoscopic Findings
19	M	71	R. E., 2/200 L. E., 10/200	R. E., many large hyaline deposits about nerve and in macular region; raised mass in macula with pigment about it L. E., same as right eye, except hyaline deposits were smaller; more pigment in macula
20	F	74	R. E., limited to counting fingers L. E., 20/40	R. E., large raised mass in macula; numerous hyaline deposits L. E., many hyaline deposits in macula and around disk; poor vision noted in left eye for only six months
21	F	76	R. E., 10/100+1 L. E., 5/200	R. E., dense hyaline bodies; also synchysis scintillans L. E., many hyaline bodies about disk; raised mass in macula
22	F	70	R. E., 4/200 L. E., 20/30—2	R. E., large area of pigment displacement and new connective tissue in macula raised 2 diopters L. E., large shiny hyaline bodies in macula and elsewhere in fundus
23	M	64	R. E., 20/100 L. E., 20/70	R. E., mass in macula raised 2 diopters; many small but dense hyaline bodies L. E., hemorrhage out from disk with edema extending to macula; dense hyaline bodies in macula
24	M	69	R. E., limited to counting fingers at 2 feet (1.1 cm.) L. E., 20/40	R. E., gray mass in macula; recurrent hemorrhages about it; large and dense shiny hyaline deposits all over fundus L. E., very dense hyaline bodies; several hemorrhages about macula
25	M	49	R. E., 20/100 L. E., 20/40	R. E., raised gray area in macula; many hyaline deposits deep in retina in macular region L. E., many hyaline deposits in macula
26	M	85	R. E., 10/70 L. E., 10/70	R. E., dense hyaline deposits, edema and pigment deposits in macula L. E., hyaline deposits; raised mass in macula
27	M	71	R. E., 20/50 L. E., 20/50	Dense hyaline deposits and disk-shaped elevated mass in both maculas; marked angiosclerosis

arteriosclerosis in most of their cases, were inclined to believe that some type of vascular degeneration in the retina was the primary cause.

And yet, if one goes over the few cases in which histologic examination has been made, a fairly strong body of evidence is found pointing to an entirely different pathogenesis. Kuhnt and Junius cited the works of Michel, Pagenstecher and Axenfeld, who described marked changes in the lamina vitrea. In Michel's case the membrane was immensely

24. Junius, P., and Kuhnt, H.: *Die scheibenförmige Entartung der Netzhautmitte (Degeneratio maculae luteae disciformis)*, Berlin, S. Karger, 1926.

thickened with large excrescences resembling hyaline deposits. In Axenfeld's case a definite break in the lamina vitrea was seen with vessels growing through it from the choroid. Behr²⁵ found degenerative changes in the lamina vitrea with a number of vessels growing through it into the choroid. He expressed the belief that the primary cause of the condition was a senile or nutritional defect in the elastic system of the choriocapillaris and lamina vitrea, which allowed a transudate to collect beneath the pigment epithelium. From this the connective tissue developed by proliferation of pigment epithelium and growth of new vessels from the choroid. Paul,²⁶ Wölfflin,²⁷ Vogt,²⁸ Rintelen,²⁹ Braun³⁰ and Verhoeff and Grossman³¹ all found defects of varying size in the lamina vitrea with, as a rule, other signs of degeneration. Only Hanssen,³² Elschnig³³ and Seefelder³⁴ found the lamina vitrea to be intact, and of these Hanssen found other degenerative changes in the membrane which caused him to agree with Behr's theory. The membrane was intact in the third case of Verhoeff and Grossman, but this was not a typical case. Most of the authors who found such changes expressed the belief that the hemorrhages so commonly observed are secondary to the degenerative changes described, arising only when new thin-walled vessels had grown into the mass from the choroid. Verhoeff and Grossman, however, stated that hemorrhage in the choriocapillaris is the primary phenomenon, causing rupture of the lamina and the formation of a transudate in front of it. What part the pigment epithelium plays in the process has been a subject of debate. Braun, for example, expressed the belief that the connective tissue mass is derived entirely from the choroid, while Rintelen and others have apparently shown that the pigment epithelium may proliferate to form masses of spindle cells such as are found beneath the retina. The retinal changes are now generally admitted to be secondary. Only one observer, Seefelder, has seen a retinal vessel enter the mass directly.

We are able to show sections which give added evidence that the primary change is in the lamina vitrea rather than in the vascular

25. Behr, C.: *Ztschr. f. Augenh.* **69**:1, 1929.

26. Paul, cited by Rintelen.²⁹

27. Wölfflin, E.: *Klin. Monatsbl. f. Augenh.* **88**:567, 1932; *Arch. f. Augenh.* **117**:33, 1926.

28. Vogt, A.: *Ztschr. f. Augenh.* **87**:173, 1935.

29. Rintelen, F.: *Ztschr. f. Augenh.* **92**:306, 1937.

30. Braun, R.: *Arch. f. Augenh.* **110**:535, 1937.

31. Verhoeff, F. H., and Grossman, H. P.: *Pathogenesis of Disciform Degeneration of the Macula*, *Arch. Ophth.* **19**:467 (March) 1938.

32. Hanssen: *Klin. Monatsbl. f. Augenh.* **95**:257, 1935.

33. Elschnig, cited by Kuhnt and Junius.²⁴

34. Seefelder, R.: *Arch. f. Ophth.* **120**:139, 1928.

system. The case was not of the typical senile variety or sections would not have been available.

Mrs. J. H., aged 29, had noticed poor vision in the left eye five months before one of us saw her. Vision was 20/15 in the right eye and 2/200 in the left eye. The peripheral fields were normal, but the left central field showed a large absolute central scotoma. The right fundus was normal. The left fundus is shown in figure 5. The large brownish gray mass in the outer part of the macular area was raised 1 diopter. With the Friedenwald ophthalmoscope it appeared solid, and what appeared to be a hemorrhage could be seen beneath the retina at the outer border of the mass. A period of observation was advised, but when after several weeks the referring ophthalmologist reported further growth of the mass and an increase in the scotoma, it was agreed that enucleation was the safest procedure.

Sections showed an essentially normal anterior segment. In the macular area was a flattened mass measuring 2 to 3 mm. in extent. This was seen to consist in part of the thickened retina containing large areas of cystic degeneration and of a layer of closely packed fibrillar spindle cells situated between the retina and the lamina vitrea. This was thickest beneath the fovea and became thinner



Fig. 5 (Mrs. J. H.).—Juvenile form of disciform retinopathy.

toward the periphery. Normal pigment epithelium was present only at the periphery of the mass, while in the area of the mass detached pigment cells were seen, some of which had assumed the form of spindle cells. There seemed to be no doubt that much of the mass was formed by proliferation of the pigment epithelium. The lamina vitrea varied in thickness under the mass, being extremely thin in places and showing one minute and one larger defect (fig. 6). Through the latter a vessel lined only with endothelium was seen to pass from the choriocapillaris into the mass, communicating with other spaces in the mass containing red cells. The retina over the mass showed absence of rods and cones, while peripheral to it these cells showed various stages of swelling and distortion. The external limiting membrane was irregular and in part absent over the mass, the newly formed tissue being here directly adherent to the neuroepithelium. There were cysts in the outer molecular layer and marked distortion of the nuclear layers. The choroid showed some accumulation of small lymphocytes beneath the mass but was otherwise normal. The retinal and choroidal vessels showed no sclerotic changes. Cystic changes were present in the retina near the ora serrata, and the same small hyaline deposits of the lamina vitrea were present in this area.

Little mention has been made in the literature of the association of hyaline deposits with central disk-shaped retinopathy. They have been

found, however, in sections by Michel, Verhoeff, Behr and Rintelen and are mentioned as being present in a few clinical cases. If one were to include cases in which hyaline deposits of any kind were present the association we have described might be most simply explained as the coincidence of different senile changes. We included, however, only cases in which there were such extreme aggregations of deposits as those which caused retinopathy previously described. The high incidence of such changes in cases of central disk-shaped retinopathy, 9 out of approximately 44, would seem to form only one indication of degenerative changes in the lamina vitrea, as part of the hyaline degeneration



Fig. 6 (Mrs. J. H.).—Microscopic section showing a defect in the lamina vitrea.

described by Rintelen. The membrane varied greatly in thickness, as seen in sections of the eyes in 1 case of the juvenile form. Evidently complete defects in the membrane may not be necessary for the production of the typical condition but only an altered permeability which permits fluid from the choriocapillaris to collect beneath the pigment epithelium.

Proliferation of the pigment epithelium seems to result from such a transudation. If vessels from the choroid do not grow into the resulting mass, hemorrhages may be absent from the picture. This was noted in several of our cases associated with hyaline deposits, as it happened,

the central plaques being rather smaller than in the average case and the visual destruction somewhat less.

A third type of retinopathy associated with changes in the lamina vitrea is that known as angioid streaks, associated in many cases with pseudoxanthoma of the skin. The reasons for considering this as a systemic degeneration of the elastic tissues need not be discussed here, except to mention the recent report of Hagedoorn,³⁵ who has apparently shown that the ophthalmoscopic picture of the streaks corresponded in his case to clearcut defects in the lamina vitrea, as reconstructed from serial sections. We simply wish to present a case in which angioid streaks and pseudoxanthoma elasticum were associated with hyaline deposits and central retinopathy.

Thelma C., a part-blooded Negro woman of 29, had always had poor vision. It had been imperfectly corrected by glasses for myopia of about 3 diopters. With best correction vision in the right eye was 20/50 and in the left eye 20/100.

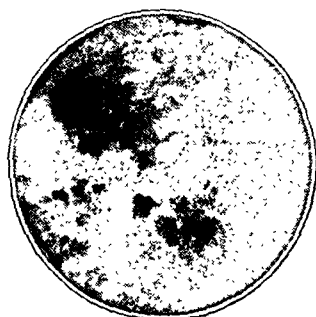


Fig. 7 ((Thelma C.).—Angioid streaks in the retina.

Pseudoxanthoma elasticum of the axillas and neck had been noted since childhood. Typical angioid streaks were present about the disks, varying in size from fine lines to broad streaks, one of which in the left eye was twice the width of the largest retinal vein (fig. 7). In each eye there was an irregular broad white streak running from the disk to the macular region. This was in the same level as the angioid streaks, and in the right eye one of the larger angioid streaks was seen in the middle of the white line extending part way to the macula. The white streaks were seen to break up into a network of finer lines in each macular region. In each macular region there was also a mass of fine white dots, closely packed together, resembling hyaline deposits. In the right eye a larger shiny deposit was present below the disk. These deposits do not show well in the photographs of the fundus, and we have been unable to locate the patient for further study. The white lines from the disk to the macula gave the impression of large defects in the lamina vitrea, partly filled by connective tissue from the choroid.

SUMMARY

Hyaline deposits of the lamina vitrea, known as drusen, may, when especially large or densely packed, cause a form of central retinopathy.

35. Hagedoorn, A.: Angioid Streaks, *Arch. Ophth.* **21**:746 (May) 1939.

This is due apparently to pressure on the visual cells or to slight changes in their locations. Hence the visual damage is usually much less than that due to primary cystic degeneration of the retina. The picture is the same whether it occurs in the hereditary form known as Tay's or Doyme's guttate choroiditis or in the form affecting isolated members of a family. The conception of a senile or presenile degeneration of the membrane fitting into Treacher Collins' group of abiotrophies seems to explain the condition as well as any other.

Such deposits are to be distinguished from Gunn's dots which, whatever their nature, are located in the inner layers of the retina and cause no visual disturbance.

Dense aggregations of hyaline deposits in the macular area are associated with central disk-shaped retinopathy in a larger proportion of cases than is to be explained by mere coincidence.

This association is taken to indicate one manifestation of more extensive degenerative changes in the lamina vitrea which seem to form the most important cause of the changes observed clinically.

A case of angioid streaks is reported associated with central retinopathy and hyaline deposits.

DISCUSSION

DR. F. H. VERHOEFF, Boston: I did not hear the essayists refer to the paper by Dr. Grossman and me on disciform degeneration of the macula, presented before this society two years ago. I think we definitely established the fact that the cause of the typical senile type of this condition is hemorrhage beneath the pigment epithelium, and we brought forward strong evidence that the so-called juvenile type of the disease is due to serous exudate beneath the pigment epithelium. From the slides shown by the essayists I cannot determine the size and exact character of the macular lesion in this case. Apparently there was simply proliferation of the pigment epithelium with formation of many spindle cells and no formation of hyaline tissue. Such proliferation is not uncommon elsewhere than in the macula, and in my opinion does not constitute disciform degeneration. In our paper it was pointed out that senile degenerative changes of the pigment epithelium no doubt predispose to hemorrhage from the choriocapillaris with resulting disciform degeneration. In the cases of disciform degeneration previously reported, in which the tissue has been examined microscopically, one of the most notable features has been the slight extent to which the lamina vitrea has been affected.

DR. ARTHUR J. BEDELL, Albany, N. Y.: (Several photographs of fundi were shown.) I should like to call attention to the importance of differentiation between exudate, deep hemorrhages and a malignant lesion. I am convinced that many eyes are enucleated which might be saved if serial photographs were made and correctly interpreted. Macular degeneration similar to that found in the presence of angioid streaks is not an uncommon finding. I have presented evidence to uphold my contention that the degeneration and the streaks are separate lesions.

DR. ARTHUR M. YUDKIN, New Haven, Conn.: This paper gives me an opportunity to express myself on a finding I believe a number of ophthalmologists have made with reference to deposits, such as hyaline deposits of drusen, in patients under 40 years of age after they have been under observation for years. Invariably when they are questioned about their early life I find that they have had quite a stormy career as far as nutrition was concerned in their first years; they will say they were fed on all sorts of formulas until the right ones were found, and they are often "Mellon's food babies." It would be well for the ophthalmologists who are making such beautiful pictures to bear that in mind and include in the histories of these cases a statement as to the early nutrition.

DR. SANFORD GIFFORD, Chicago: I am sorry Dr. Verhoeff did not hear me say that I would let him speak for himself. I knew from reading his paper that he had a different opinion, and I thought that if I discussed his paper he would want to discuss it over again. As a matter of fact, there were ruptures in the lamina vitrea in 2 of Dr. Verhoeff's 3 cases. The only question is whether the hemorrhage was a primary factor, as he thought, or whether it was secondary. In the case in the early stage there was no rupture in the lamina vitrea, but there was exudate. According to Behr, something happens to the lamina vitrea which is not necessarily a rupture; fluid gets in front of it, and if there is a definite defect, the vessels grow from the choroidal capillaries. There is only one man who has seen one of the retinal vessels growing into a mass, and that is Seefelder; usually the hemorrhages come from the choroidal vessels. I do not want to confuse the fact that I am talking about two different conditions. I am talking about central degeneration due to hyaline deposits alone, and secondly about the association of hyaline deposits with disk-shaped retinopathy of Kuhnt and Junius. I was also talking about one other thing which I did not have time to discuss and which I did not want to start an argument about with Dr. Verhoeff, and that is angioid streaks. There is a recent article by Hagedoorn which seems to show that these angioid streaks are ruptures in the lamina vitrea. It was published recently in the ARCHIVES (21:746 [May]; 935 [June] 1939), and I think that there is some relationship between ruptures and angioid streaks.

I think I also mentioned Dr. Bedell, and I should say his pictures in his atlas are the only ones I could find in the American literature of this fundus picture.

In answer to Dr. Yudkin, I personally do not think that original nutritional factors are important in this condition because of the hereditary history which occurs in Tay's familial choroiditis. There was certainly nothing in these cases pertaining to nutrition, and I believe these isolated cases are of the same nature in all respects.

ROLE OF STATES OF ANXIETY IN THE PATHOGENESIS OF PRIMARY GLAUCOMA

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It is the contention of this paper that the eyes of some patients with glaucoma register states of anxiety just as those of other persons with glaucoma record changes in weather, poor illumination, poorly balanced diets, cardiovascular disturbances, acute febrile diseases and sudden increase of blood pressure.

This contention is based on data obtained by the study of the emotional life of patients suffering from glaucoma and on the meager data found in the ophthalmologic literature. The significance of acknowledging the existence of a relation between the "psychic" and "organic" factors in glaucoma lies in the fact that it brings one a little nearer to the understanding of the pathogenesis of this condition.

The heads under which the subject will be treated in this paper are as follows:

Report of Cases

A Review of the Disturbances of Physiologic Processes Caused by States of Anxiety and Their Anatomic Substratum

Relation Between States of Anxiety and Hypertensive Crises in Glaucoma

REPORT OF CASES

The cases reported here are all of primary glaucoma. The diagnosis has been based on careful examinations during long periods of observation. The history of the illness, the study of the size of the cornea, the depth of the anterior chamber, the size and reactions of the pupils, the biomicroscopic findings, the appearance of the optic disks and the retinal and anterior ciliary blood vessels, observations made at perimetric, scotometric and campimetric examinations and with the tangent screen and the response of the ocular tension to provocative tests (the reaction of the pupils to light, convergence and drugs) have been mainly the data on which the diagnosis has been based. For practical reasons and avoidance of repetition, none of the findings

Read before the Section of Ophthalmology of the New York Academy of Medicine, April 17, 1939.

This paper is the first of two related papers, the second of which, entitled "Psychosomatic Interrelations: Their Therapeutic Implications in Glaucoma," appears in this issue of the ARCHIVES, page 91.

elicited by the routine examinations was mentioned in individual case histories.

An attempt was made to obtain information regarding states of anxiety from the patient's life history.¹ On account of the limited space only essential data are recorded here.

CASE 1.—L. T., a garage owner, came under my observation seven years before the writing of this report, at the age of 39 years, with signs and symptoms of bilateral simple glaucoma. The left eye was operated on, and the tension remained normal without the aid of local medication. The result has remained good from every point of view. For reasons irrelevant to the subject of this paper, the right eye was not operated on. The patient consented, however, to call regularly every month for observation and advice.

The tension in the right eye could be maintained within normal limits only by using regularly local medication. In the course of the past seven years the patient found that "under normal circumstances" the instillation of drops in the right eye at 11 a. m. and 5 p. m. sufficed to prevent the appearance of rainbow rings or of any unpleasant feeling in or around the eye. On the other hand, he became convinced that the "abnormal circumstances" which brought about the feeling of tightness in the eye, cloudy vision or perception of rainbow rings were emotional upsets. He stated repeatedly that when he was on a vacation, away from business and relaxed, the eyes felt better.

In September 1935 he was subjected to an emotional shock by a death in his family. The ocular tension in the right eye was found to be 15 mm. of mercury higher than it had been for some time before, although the drops had been instilled as usual.

In January 1937 the ocular tension was found to be 36 mm. of mercury. On talking over with me the probable reasons for this ocular hypertension, the patient volunteered the information that he had had serious business worries.

On March 11, 1938, the patient stated he had noticed that within two hours after excitement he began to see rainbow rings around lights with his right eye only. Even "slight arguments" with his wife were recorded by the right eye. When listening to political speeches broadcast about enemy countries the right eye felt annoyed. Whenever he was being rushed the right eye was uncomfortable.

For years he had observed that the right eye was comfortable when the instillations were made at 11 a. m. and 5 p. m. The eye "resented" even one-half hour delay. However, when going for week-ends to his country home, he could postpone the use of drops for one to one and a half hours without feeling any discomfort. Under "normal circumstances" the eye felt no discomfort after the instillation of the drops, but after excitement he felt a cramp in his eye after using the drops. If the patient was annoyed or upset at 4:30 p. m., he was certain to see rainbow rings within two hours, even if the drops had been used at the regular time (5 p. m.). The patient found that the best way of preventing attacks was to use drops of pilocarpine^{1a} before and after business interviews or whenever he had gone through difficult situations or emotional upsets.

1. Circumstances excluded the possibility of carrying out lengthy psycho-analytic surveys of the patient's personality. But even the relatively short time of interrogating yielded a surprising amount of information. Of course, a deeper study of the patient's emotional life is likely to uncover most valuable data.

1a. Pilocarpine hydrochloride was used in this and in subsequent cases.

In this case a patient is being dealt with whose right eye (which had not been operated on) became a faithful and delicate recorder of his states of anxiety.

One may attempt to interpret this situation as "autosuggestion," and, superficially, this interpretation sounds plausible.

Past experiences, over a period of several years, have taught this patient that states of anxiety (worry, fear of explosions or of fires in his garages, labor trouble, insecurity of income and quarrels) bring about minor attacks of ocular hypertension accompanied by the usual symptoms (cloudy vision, rainbow rings and an uncomfortable feeling in and around the right eye). It is therefore logical to assume that whenever the patient is subjected to emotional stress he will expect a recurrence of these symptoms. This is not autosuggestion. It is knowledge derived from past experiences. Besides, the assumption that a rise of ocular tension is brought about by autosuggestion does not explain anything and does not bring the problem nearer its solution, because the questions immediately rise: How does autosuggestion bring about a rise of ocular tension? By what mechanism would a mental process of this kind raise the ocular tension?

CASE 2.—B. K., a housewife, came under my observation in December 1936, at the age of 43, with simple glaucoma in the left eye. (The right eye had been operated on for glaucoma some months before.) She complained of frequent attacks of headaches, "eyestrain," rainbow halos around lights and cloudy vision. These attacks had their inception seven years before the preparation of this report, about the time when her husband, who had been a drunkard for years, began to break up things in the home. The patient complained of general weakness, nervousness and mental depression and insisted that the attacks in the eyes appeared after emotional upsets. She stated that her sleep was poor, that she was often nauseated and had crying spells and that she and the children lived under a continuous dread of what might happen when the father came home in the evening. She was easily upset. In the course of time and gradually the patient unfolded the story of her life. She had been married for about twenty years and was sexually frigid. The marital relations were never good. Her husband's reaction to her sexual deficiency at the beginning was to get drunk only occasionally; later he did so frequently, and the more he behaved "shamefully" the more frigid the wife became. Here was a situation which was much too involved for me. A psychotherapist with a solid medical knowledge might have been able to straighten out conditions. But there was no inclination to cooperation on either the husband's or the wife's side. Even pharmacologic treatment could not be carried out punctually. Surgical intervention was refused, since the operation on the first eye had been far from successful.

CASE 3.—L. D., an engineer-inventor aged 63, complained that for the past year he had attacks of blurred vision, rainbow halos and dull pain around the right eye. Two years previously, only a few months before the appearance of symptoms of glaucoma, the patient had an accident which gave him a broken knee and concussion of the brain.

A diagnosis was made of subacute glaucoma in the right eye and simple glaucoma in the left eye.

The tension in the right eye was —65 mm. and in the left eye 13.5 mm. Applications of ice and frequent instillations of a 2 per cent solution of pilocarpine, reduced the tension in the right eye to 17 mm. of mercury within one day.

The history of the relation between his states of anxiety and his attacks of ocular hypertension is illuminating.

On the evening of Sept. 23, 1937, the patient had a five hour conference with the promoters of his inventions and became extremely nervous. The right eye, which had felt perfectly well during the entire summer, began to trouble him that night. The next morning the tension of this eye was 60 mm. of mercury, which, however, receded to 16 mm. within two hours with applications of ice and frequent instillations of prostigmine and mecholyl.

On September 30 the patient reported that when working on blue prints, calculating, designing and thinking hard the right eye felt "poorer."

On Jan. 24, 1938, he informed me he had repeatedly observed that if he managed to get a good night's sleep the attacks of ocular discomfort disappeared completely. He knew that excitement was doing harm to his eyes (he became easily excited) and that whenever he succeeded in controlling his temper and in avoiding excitement he could prevent the attacks.

In May 1938 he stated that since he had been doing "brain work" the entire day he was nervous and easily excitable. Once after having worked continually the entire day, he began "to see smoke and rainbows." When he was nervous (he said on September 6) he "felt tension in his eyes."

On September 16 he had to wait at the office for some time, which seemed unduly long to him; he became very nervous, and when the tension was measured it was 72 mm. of mercury in the left eye. Within one and a half hour the tension was brought down to 16 mm. by the aid of instillation of pilocarpine and applications of ice.

On November 10 the patient made the characteristic statement that up to several months previously he was constantly worried that his small capital would not last until his inventions began to bring in returns. A short while later he lost his house and his money; he then felt freed of this state of uncertainty. Ever since, his mind had felt relieved and his eyes were better.

He summarized the relation between states of anxiety and the attacks in his eyes in the following manner: The attacks appeared (1) when he was worried or mentally upset and (2) when he was doing hard mental work continually for many hours.

CASE 4.—S. N. came under my observation nineteen years previous to the writing of this report, at the age of 42; he is a writer on political matters, highly cultured and intelligent.

A diagnosis of bilateral simple glaucoma was made.

The left eye was operated on successfully, and the tension remained normal without the use of medication for eighteen years. The right eye, though operated on, did not maintain a normal tension without the use of pilocarpine. This eye recorded occasionally emotional upsets and states of anxiety. During the period of nineteen years the patient repeatedly made the statement that slight attacks of blurriness and rainbow halos occurred in the right eye after excitement, unpleasant business interviews, worry, "hard mental work" and states of fatigue and exhaustion which, as is well known, are accompanied by mental irritability. On having to meet people he disliked or on hearing bad news, he had pain in his temples.

CASE 5.—J. R., a paper bag manufacturer, came under my observation when he was 50 years of age. One month before his first call he had had a subacute attack of glaucoma in his right eye, which yielded somewhat to the half-hearted use of pilocarpine. Examination revealed bilateral simple glaucoma. The right eye, which showed signs of serious damage to the visual field, was successfully operated on; the left eye was treated by the frequent use of drops and applications of ice. The patient's personality and states of anxiety were also surveyed. It took several months before the tension in the left eye descended and remained at a safe level (around 20 mm. of mercury) under the influence of drops, used only twice a day.

The emotional background of this patient succinctly stated is as follows: Two and one-half years before consulting me his business began to recede and he was losing money rapidly. This worried him so much that he could not sleep. In addition, he followed with great interest the foreign political news, which were threatening at the time. This upset him daily for several months. He felt his eyes become harder, throbbing and painful whenever he was excited.

After the operation on his right eye, he concentrated all his attention on that eye. Although his visual acuity was better than before the operation, the tension staying normal and the eye showing no signs of irritation, he kept on worrying about this eye. He also began to have attacks of general weakness, dizzy spells and an annoying feeling of tightness in the muscles of his shoulders. Careful and repeated examinations by various clinicians led to the diagnosis of nervous exhaustion, following a state of anxiety of long duration.

It is noteworthy that persistent, frequent local treatment of the left eye for three to four months finally succeeded in reducing the ocular tension to a normal level and later in keeping it there for a whole year, drops being used only once or twice daily.

It is my impression that the lowering of the ocular tension in the left eye took place mainly because the tangled financial conditions improved and the patient was gradually made to realize that mental relaxation and avoidance of states of anxiety were essential for the recovery of his eyes.

CASE 6.—S. G., a housewife aged 61, had bilateral glaucoma simplex. She had gone through a great deal of trouble. Her husband had been an invalid for twelve years. Of five children, two were seriously ill. She came under my observation in July 1937. During the first two months of the period of observation the tension remained 13 to 16 mm. in the right eye and 13 to 17 mm. in the left eye. Only on one occasion was the tension found to be 24 mm. in the right eye and 22 mm. in the left eye.

At the end of August 1937 she lost her husband and a few months later, a daughter. She had to move to the son-in-law's home and take care of an orphan. She cried continually and was extremely unhappy. After this, the tension began to rise gradually and settled at a higher level: 26 to 28 mm. in the right eye and 22 mm. in the left eye. A more frequent and intensive medication could not bring the tension back to the previous lower level, and the circumstances were such that nothing could be done to allay her state of anxiety.

CASE 7.—B. R., a woman aged 42, came under my observation on June 24, 1938, with bilateral simple glaucoma of about four years' duration.

On taking the history of her emotional life, it was revealed that she had had serious disagreements and quarrels with her husband ever since she was married

(twenty years previously). Her sleep had been poor for years. Before the glaucoma appeared her husband had lost all his money, and she helped him recover some by working sixteen hours a day. Her vision frequently became cloudy, and she saw rainbow halos after excitement, mental upsets and crying spells. Similar attacks occurred before menstruation and before a change of weather. On listening to alarming radio news she usually would become upset and cry. She found that she could "stop" the attacks by taking a walk for one-half to three-quarters hour and diverting her thoughts from the unpleasantness at home.

CASE 8.—S. C. D., a lawyer, came under my observation on Feb. 18, 1927, at the age of 44 years. A diagnosis of presbyopia was made.

In 1934 and during the years following up to the present, the patient has had at infrequent intervals (every few months) attacks of blurred vision accompanied by rainbow rings around lights and some pain. The diagnosis was bilateral simple glaucoma.

On Jan. 21, 1939, he reported the observation that undue excitement brought about the attacks in his eyes. (Playing bridge for a few hours also was followed by this sort of crisis.)

A survey of this patient's emotional upsets revealed: 1. He was of a worrisome disposition and was always afraid of something that never happened. This state of anxiety was intensified by his financial collapse (1929), and it remained constant. 2. His sister was demented and gave him a great deal of trouble. His wife informed me that whenever the telephone rang the patient became pale and trembled for fear something might have happened to the sister. 3. The wife was extremely verbose; more or less, verbal skirmishes were taking place daily.

The last attack of "rainbows" and cloudy vision took place after he received a telephone message about the sister. This attack lasted four hours in spite of the fact that he had used drops of pilocarpine immediately at the beginning of the crisis.

CASE 9.—B. B., a salesman, came under my observation in October 1936, at the age of 57. A diagnosis of bilateral glaucoma simplex was made.

The complaint was that for the past three years he had been seeing rainbow rings around lights, occasionally having pain in the eye, and that vision invariably blurred when he played cards for several hours. The trouble would appear in both eyes during the excitement of card playing; when he did not play cards only the left eye was subject to occasional blurred vision but no rainbow rings. The symptoms persisted until the ocular tension was permanently reduced by a trephining operation.

CASES 10 and 11.—The history of the following two cases, reported by Seidel,² illustrates how anxiety may affect the eyes in the early stage of glaucoma. A woman aged 58 and a man of 59 were admitted to the hospital for observation and treatment. Each had chronic glaucoma in one eye only. The other eye did not show evidence of any trouble. The dark room test, used repeatedly on each patient before the admittance to the hospital, had consistently given negative results. During their stay in the hospital these two patients were seriously frightened. One of them was led to believe by a misunderstanding that bad news had arrived from home; the other patient was told mistakenly by one of the

2. Seidel, E.: Ueber die psychische Beeinflussung des intraokularen Flüssigkeitswechsels, *Ber. ii. d. Versamml. d. deutsch. ophth. Gesellsch.* 49:336-339, 1932.

younger assistants at the clinic that an urgent operation was necessary on the good eye. The fright was not followed by an ocular crisis, but the dark room test the next day elicited, for the first time, a marked increase of ocular tension in the eye which had previously been negative to the dark room test. This appeared after one hour's stay in the dark and returned to normal at the end of one-half hour's exposure to daylight. This positive response was found only once in the woman but for six consecutive days in the man. Subsequent examinations during the following two years showed no rise of pressure to the dark room test.

These two observations are remarkable not only because of the light they throw on the early phase of glaucoma but because they demonstrate that states of anxiety may shift the anatomic and physiologic mechanism in the eye in such a way as to bring about a positive response to a diagnostic test for glaucoma.

An interesting case of psychogenic ocular hypertension is also reported by Krasso.³ The paper should be read in the original.

The 11 cases here reported should be sufficient to throw into relief the possibility that in some glaucomatous patients there may be a definite relation between states of anxiety (worry, fear, fright, sorrow, disappointment, despondency, self reproach and many other states of awareness of distress too vague to be expressed in words) and recurring crises of ocular hypertension.

Some of the reports are fairly convincing; some may not appeal at all, especially to those who have never investigated such matters. It will take hundreds of reports made by a large number of clinicians to clear up the subject somewhat.

A REVIEW OF THE DISTURBANCES OF PHYSIOLOGIC PROCESSES CAUSED BY STATES OF ANXIETY AND THEIR ANATOMIC SUBSTRATUM ⁴

Physiologic experiments and clinical observations have established that states of anxiety cause a widespread and more or less intense disturbance of the physiologic processes throughout the entire body.

Some of the effects of states of anxiety are:

- (a) Increase of the liberation of epinephrine
- (b) Increase of cardiac output
- (c) Shortening of the blood clotting time
- (d) Changes in blood pressure, in metabolic rate and in the distribution of the blood to the different parts of the body

3. Krasso, I.: Ueber den psychischen Einfluss auf die Entstehung von intrakularer Drucksteigerung, *Ztschr. f. Augenh.* **70**:336-339 and 355-384, 1930. (This paper contains a good bibliography.)

4. (a) Best, C. H., and Taylor, N. B.: *The Physiological Basis of Medical Practice*, Baltimore, William Wood & Company, 1937. (b) Rosett, J.: *The Mechanism of Thought, Imagery and Hallucination*, New York, Columbia University Press, 1939. (c) Cannon, W. B.: *Bodily Changes in Pain, Hunger, Fear and Rage*, New York, D. Appleton and Company, 1920.

- (c) Increase in the number of red blood cells
- (f) Increase of the cardiac and respiratory rate
- (g) Hyperglycemia
- (h) Arrest of digestion
- (i) Dryness of the mouth and throat
- (j) Tendency to frequent urination or diarrhea
- (k) Tremors
- (l) Muscular tension
- (m) Pallor or flushing of the face
- (n) Disturbance of attention, memory,⁵ reasoning and judgment and probably many others

The ocular disturbances occurring during acute states of emotion are :

- (a) Increase in the size of the pupils
- (b) Enlargement of the interpalpebral fissure
- (c) Bulging of the eyes
- (d) Hypersecretion of the lacrimal glands
- (e) Spasm of retinal arteries (in older patients), and probably many others

Most of these manifestations are similar to those appearing after a sudden and large increase of epinephrine in the blood stream (Cannon ^{4c}).

The structures which have a relation to the emotional disturbances set going in the organism are the thalamus, the hypothalamus, the cerebral cortex (which constitute the highest centers of the autonomic nervous system) and the endocrine glands, especially the adrenal and the pituitary glands.

Rosett ^{4b} summarized the physiologic anatomy of the mechanism of emotions as follows :

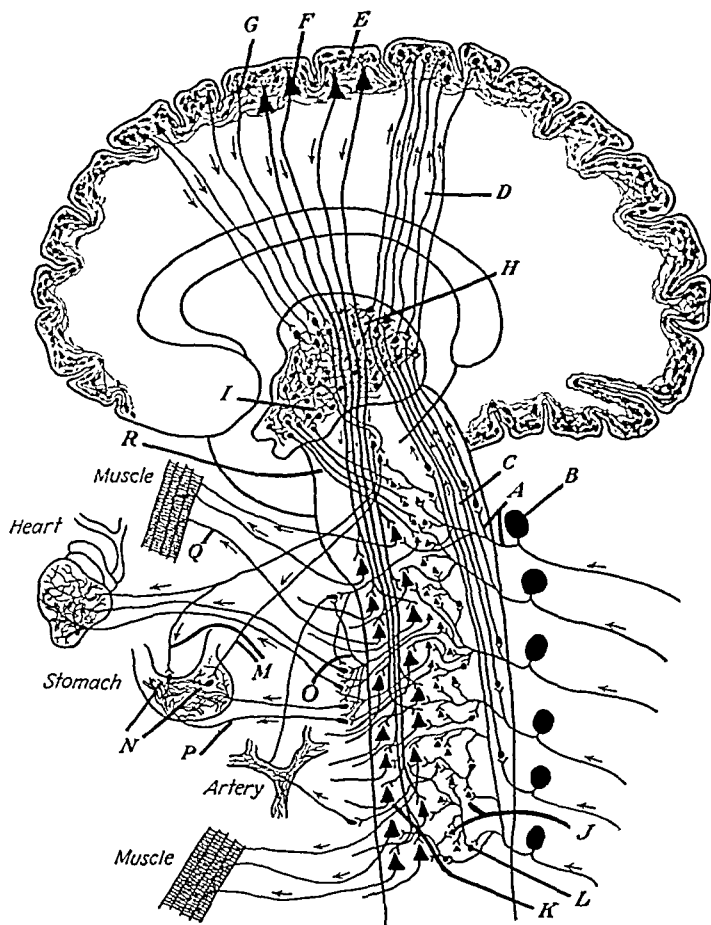
The nerve fibers which convey sensory impulses from the receptor organs end in branches in connection with a number of cells in the thalamus. Some of these cells, in their turn, send out nerve branches which end in certain small areas in the cerebral cortex. Other branches of these cells, however, establish communication with cell groups in the hypothalamus, and the cells of the latter send out nerve fibers which are connected with the complicated system of nerves and nerve relay stations which innervate the blood vessels of the entire body, all the glands and all the internal organs. It is this latter system of nerves which is immediately responsible for the bodily changes which take place in the emotions [and especially in states of anxiety].

In chronic states of anxiety, impulses are being sent out from the higher centers of the autonomic nervous system (in the thalamus and hypothalamus) and produce widespread bodily disturbances similar to those observed in acute emotional upsets (terror, anger and similar

5. Fremont-Smith, F.: The Influence of Emotional Factors upon Physiological and Pathological Processes, read before the New York Academy of Medicine, Dec. 2, 1938.

emotions), the difference being only a matter of degree of intensity. Loss of appetite and of sleep, impairment of clear thinking, pallor of the face, disturbance of menstruation and of digestion, impairment of energy in general and recrudescence of some diseases testify that bodily changes take place also under the influence of chronic states of anxiety.

The pituitary gland secretes a number of powerful hormones which are conveyed by means of the circulation to the highest centers of the autonomic system in the thalamus and hypothalamus and to the organism as a whole.



A schema of the lateral view of the cerebrospinal and autonomic nervous systems. *A* indicates the afferent (entering) spinal nerve; *B*, the spinal ganglion; *C*, the sensory pathway; *D*, the thalamocortical nerves; *E*, associated systems; *F*, the corticospinal (pyramidal) pathway; *G*, corticothalamic nerves; *H*, the thalamus; *I*, the hypothalamus; *J*, the lateral column of autonomic cells; *K*, the ventral column of motor cells to the skeletal muscles; *L*, an intercalated nerve; *M*, the pre-ganglionic fiber of the upper part of the autonomic nervous system; *N*, the postganglionic fiber of the upper part of the autonomic nervous system; *P*, the postganglionic fiber of the middle autonomic nervous system; *Q*, a motor nerve to the skeletal muscle, and *R*, the visceral pathway from the hypothalamus. (From Rosett, J.: *The Mechanism of Thought, Imagery and Hallucination*, New York, Columbia University Press, 1939.)

It is well known that in states of emotional stress there is a marked increase of epinephrine-like substances in the blood and tissues, originating in the adrenal medulla and at the sympathetic nerve terminals. The secretory function of the medullary portion of the adrenals is under the control of a nucleus situated in the floor of the fourth ventricle, and this nucleus is in direct anatomic relation with the highest regulating centers of the autonomic nervous system.

To summarize:

(a) States of emotion are evolved in the same regions of the brain in which are located the highest centers of the autonomic nervous system.

(b) The adrenal and pituitary glands are functionally connected with the autonomic nervous system and participate in the activity of the mechanism of emotions.

(c) The effects of emotions are transmitted throughout the entire organism by means of the autonomic system and the hormones of the pituitary and adrenal glands.

RELATION BETWEEN STATES OF ANXIETY AND HYPERTENSIVE CRISES IN GLAUCOMA

The previous section has indicated that the autonomic nervous system and the endocrine glands reach every organ and tissue in the body, the eyes, of course, not excepted.

The question which constitutes the principal topic of this paper is whether states of anxiety affect glaucomatous eyes in a somewhat different manner than normal eyes, and, if so, in which way?

There is much evidence to show that (a) the two parts of the autonomic nervous system (the sympathetic and parasympathetic) are out of balance within the glaucomatous eyes^{6c} and that (b) states of anxiety have a different effect on glaucomatous eyes than on normal eyes.

Some evidences for (a) are:

1. The power of accommodation is diminished, and there is a tendency for the pupils to be somewhat dilated (underaction of the parasympathetic system).

6. (a) Inman, W. S.: Emotion and Acute Glaucoma, *Lancet* **2**:1188-1189, 1929; Emotion and Eye Symptoms, *Brit. J. Psychol. (M. Sect.)* **2**:47-67, 1921; The Emotional Factor in the Causation of Diseases of the Eye, *Tr. Ophth. Soc. U. Kingdom* **55**:423-434, 1935. (b) Sussman, R.: Psyche und Auge, *Klin. Monatsbl. f. Augenh.* **81**:103-109, 1928. (c) Lagrange, H., in discussion on Bidault, R.: Le traitement médical général du glaucome, *Bull. Soc. d'opht. de Paris* **49**:446-507, 1937; La sympathose glaucomateuse (remarques sur la crise de glaucome), *Bull. Acad. de méd., Paris* **120**:234-238, 1938; Douleur viscérale, sommation glaucomateuse, *Arch. d'opht.* **49**:741-742, 1932. (d) Traquair, H. M.: Glaucoma, with Special Reference to Medical Aspects and Early Diagnosis, *Brit. M. J.* **2**:933-938, 1935.

2. A solution of epinephrine hydrochloride (1:1,000) dilates the pupil when instilled in the fornix of a glaucomatous eye which is not under the influence of a miotic, but it does not dilate the pupil in a normal eye of a normal person.

3. Pupils of glaucomatous eyes usually need a stronger solution of pilocarpine for contraction (showing that the parasympathetic innervation of the sphincter pupillae is hypotonic).

4. Atropine in usual doses increases appreciably the intraocular pressure in the glaucomatous eye; the intraocular pressure of the normal eye is not increased by ordinary doses of atropine.

5. Ordinary doses of pilocarpine and physostigmine salicylate reduce appreciably the ocular tension in a certain group of glaucomatous eyes, while the tension in normal eyes remains unaffected.

6. Drugs with a specific action on the autonomic nervous system have also a specific action on glaucomatous eyes.

7. Emotional upsets, febrile diseases and sleeplessness, which have a strong unbalancing influence on the function of the autonomic nervous system, also have a tendency to increase the ocular tension.

As evidences for *b*, proving the effect of states of anxiety on glaucomatous eyes, one could mention the following facts:

1. The cases reported in the first section of this paper present such evidence.

2. The ophthalmologic literature contains reports (vague and meager, though they are) of attacks of acute glaucoma following violent emotions.⁵

3. There is scarcely an ophthalmologist of experience who has not encountered some cases of glaucoma in which a recrudescence of the condition was not attributable to states of anxiety.

4. Many patients, when permitted and encouraged to talk, have described minor or major attacks of glaucomatous crises during or following states of anxiety.

5. There is no case on record of an attack of glaucoma in a normal eye following an emotional upset.

CONCLUSIONS

In a certain percentage of cases states of anxiety act as a precipitating factor for the development or maintenance of a high intraocular pressure in glaucomatous patients.

Some of the glaucomatous crises are therefore preventable by proper attention to the patient's emotional life.

DISCUSSION

DR. SMITH ELY JELLIFFE (by invitation): 'Tis a maxim of ancient lineage that "fools rush in where angels fear to tread," but as what rushing I may indulge in is limited to ten minutes, the episode will not be very exciting, especially as I would bring old time thoughts with me. What the Homeric Greeks may have thought of glaucoma I do not know. They evidently knew it and named it. Certainly by the time of Socrates a true psychosomatic monism was fairly entrenched in medicine, at least for the Scythians if not for the Greeks. These outsiders, as Socrates related, believed that without a consideration of the soul, i. e., the body as a whole concept, no real insight could be gained into many of the problems of medicine.

A present day dweller at Harvard, the Modern Athens (at least many at Harvard are likely to consider it the intellectual Acropolis of this continent), has expressed the thought explicitly after stating that the Pilgrim Fathers of the modern scientific imagination were these ancient Greeks. Whitehead, of Harvard, in his "Science and the Modern World" has put it this way:

"The concrete enduring entities are organisms, so that the plan of the whole influences the very characters of the various subordinate organisms which enter into it. In the case of an animal, the mental states enter into the plan of the total organism and thus modify the plans of the successive organisms until the ultimate smallest organisms, such as electrons, are reached. Thus an electron within the living body is different from an electron outside of it, by reason of the plan of the body, and this plan includes the mental state."

Naturally the fact is accepted without much emphasis that there are always gross or minor accidental happenings that might interfere with the serenity of this general point of view, but one is here reminded of the principle of psychosomatic monism, which I take it is the central theme of Dr. Schoenberg's paper. What in the Whitehead quotation is designated as mental state is partly restated in Dr. Schoenberg's use of the term anxiety.

The ophthalmologists here are much better equipped than I to discuss the innumerable factors of biochemical, physiologic and pharmacologic import in the eye microcosm, which in glaucoma is reflecting some type of disturbance of the wisdom of the body.

The fragment which I might bring to the discussion would emphasize the need for more precise formulations of the term "anxiety." In thermodynamics one discusses the action of heat units on foreign bodies. In order to be of any service, such deliberations must get down to actual heat units from absolute zero to the melting temperature of platinum or one gets nowhere. Anxieties are dynamic energetic situations and not a static abstraction. Every tub stands on its own bottom.

I am not prepared to state that there are as many grades of anxiety tensions as there are of heat on the thermodynamic scale, but it is imperative to bear in mind that "anxiety" as an abstraction is of little service in actual understanding, even if it may be a verbal symbol for a warning "stop, look and listen."

Neither can the psychoanalytically oriented psychiatrist be entirely satisfied with the conscious manifest descriptions of anxiety, such as are well portrayed in the case of histories offered. There are always some things deeper and more specific in the general symptoms that go by the name of anxiety than appear behind the front of economic distress (case 1), alcoholic husbands (case 2), patent right rivalries of the inventor (case 3), worry (case 4), business reverses (case 5), husband and money troubles (case 7) and loquacious wives (case 9).

This audience does not need to be reminded that one of those Pilgrim Fathers alluded to earlier, Sophocles, drew a universally present and still valid picture of a self-inflicted blindness in the case of Oedipus. Blindness is the goal reached by glaucoma. In what sense may it be discussed as a compromise reaction of frustration and aggression which utilizes a destructive regressive mechanism, bringing the individual into an intrauterine environment as black as night? Any conscious logic in such a fantastic effort at organ suicide is millions of years away.

I wonder if I should meet with apprehension if I should hazard the conjecture that the eye is the most potentially aggressive of all the senses. Is there any organ of the body that can reach out and take hold of billions of years of time and billions of miles of space? To pass from the sublime to the ridiculous, consider the small detail offered in the *New York Times* of Hitler's scores of glasses as a small contribution to the psychology of an aspect of the aggressive omnipotency wish.

If short eyesight and omnipotence (in the unconscious) are closely equated, any conflict that tends to inhibit omnipotence must have its repercussions in the bodily organs.

There are so many facets to this general problem that any restriction to a ten minute discussion brings about annoyance. Thus Dr. Schoenberg, wisely perhaps, said nothing about the general bodily configuration of his patients. Other constitutional studies have indicated that the leptosome habitus and simple glaucoma are intimately equated, while the aggressive fighter of the pyknic formation gets inflammatory glaucoma. What mental states lie behind these bodily formations?

I turn aside from the complicated problems of anxiety, inhibition and symptoms because it would lead into deeper waters than I can conveniently wade in here. There is practically no thorough analyses of the unconscious patients with glaucoma. Dunbar in her masterly collection gives some of the available facts. In a sense, perhaps, because my own initial studies on certain myopias (1926) "put me on the spot" and as some myopias and some glaucomas are equated, these early "speculations," as I called them, have been made realities. Blindness as a reversible process, as in conversion hysteria, is a story as old as Hippocrates, but what I have designated as malignant irreversible conversion processes are still in need of further study. Inman, Sussmann, Mohr and Seidel have started these individual analyses of the unconscious process of flight from reality.

I have no case histories to report. This morning a psychiatric social worker mentioned that a patient she had seen was having glaucoma, and I asked her to say a few things about it. She said, "This is a

young, charming, delightful, educated and well-to-do matron with two or three young children" who, if I may interpose a story, got into a "situation." You remember the story of the young man who followed the young lady on the street, with ideas, and as he got up to her he turned and said, "Well, do you or don't you?" And she said, "I don't, but you talked me into it." Something had happened in the life of this young matron which was dangerously near to the situation of being talked into it. And how did she respond? What was the nature of her flight? What apprehensions arose within her mental systems and what was the anxiety that arose as a symbol, as anxiety always is a symbol of danger? Emotions are nothing more or less than precipitates of ancestral experiences, and the general emotional makeup of a person in response to millions of years of ancestral experience has a specific application when a particular generalized type of emotional (namely anxiety) situation is discussed, which, as I have already indicated, is nothing but a warning on the part of the person to stop, look and listen. In other words, the instinctual impulses of which I have already spoken with regard to the little story I told are always active. They have to have some mechanism of control. The old theologians, philosophers and psychologists talked about instinct being regulated by reason, but when the problem of anxiety is discussed, one is discussing something a little more specific than the abstraction of instinct control by "reason" and/or by "conscience."

As I said, I have no case histories to report—only a few scraps, in all of which the flight mechanisms from intense aggressive id impulse assaults have overwhelmed the patient and an intense super-ego tyranny has turned extraverted aggression to introverted self destruction, i. e., a typical castration. To present even a thumbnail sketch in terms other than those of the special concepts of psychoanalysis would occupy an hour or more.

DR. JOSHUA ROSETT (by invitation): I can remind Dr. Jelliffe of another patient of his. A number of years ago he reported before the Association for Research in Nervous and Mental Disease a case of multiple sclerosis in which the disease was either caused or precipitated by prolonged emotional stress. I remember how stunned the listeners were at the report that mere emotion could be causative of so definite an organic pathologic condition. One is no longer stunned by such reports. Since that day physicians have learned to understand the relation between the emotional state and the coincident and subsequent alteration in the tissues. They now know that the essential factor underlying an emotion is an activation and, in certain cases, an over-activation, of the vegetative and fundamental functions of the body and that the normal significance of an emotion is to adjust the organism from moment to moment to the changes in his surroundings. For example, the emotional state of fright normally results in dilatation of the pupil, in an increase of the cardiac and respiratory rates, in an increased output of epinephrine and sugar, in a concentration of the attention and in a number of other events. In extreme cases these may attain pathologic proportions. Attention may become concentrated to a point where the person is practically unconscious, and the alteration in the tissues may leave permanent and baneful traces behind them, the cause of which is frequently difficult to detect.

In view of these facts, Dr. Schoenberg's paper, dealing with the effects of anxiety, apprehension and worry in determining attacks of increased intraocular pressure in glaucoma becomes entirely understandable. The cases which he cited are in themselves sufficiently convincing, and his outline of the autonomic nervous system as the physical substratum of the emotions is fully corroborative of his thesis. All that I can do by way of amplifying his thesis is to call attention to the fact that the highest controlling nerve centers of the autonomic system—in the hypothalamus—are closely related to the visual apparatus.

The hypothalamus is a much more prominent structure, with relation to the whole brain, in fishes, amphibia, reptiles and lower animals than it is in man. As a matter of fact, the human hypothalamus is only about one three hundredth part by weight of the rest of the brain. Yet, within this small structure are contained nerve centers which preside over most of the fundamental functions of the body—temperature balance, water and sugar metabolism, the rhythm of sleeping and waking and others. Experimental and clinical proof is rapidly disclosing the fact that the several nuclear masses have each a specific function. The hypothalamus contains a center for the dilatation of the pupil. The entire structure is connected by a system of complicated pathways with the optic thalamus and the cerebral cortex above, with the pituitary gland below and with the brain stem and the spinal cord behind.

The close association of the emotional mechanism of the hypothalamus with the visual apparatus becomes apparent from the fact that the superior surface and the sides of the optic chiasma are buried within the hypothalamus and that, as a matter of fact, the optic chiasma in the embryo is formed within the hypothalamus. The arterial system from the circle of Willis supplies both the hypothalamus and the optic chiasma.

But there is still much that is not known about the hypothalamus. For example, a number of its cells contain colloid inclusions, a fact which raises the suspicion that this portion of the brain is possessed of a secretory function. If such should prove to be the case, what might be the effect of this secretion on the optic pathways, which share with the hypothalamus the same blood supply?

DR. MARK J. SCHOENBERG: Because Dr. Jelliffe has mentioned Socrates, I wish to call attention to what Socrates had to say about this subject twenty-four hundred years ago: "Just as we cannot treat the eye without the head, and we cannot treat the head without the body, so we cannot treat the body without the soul." Hippocrates, who had a marvelous clinical sense, said that the Thracians were much wiser and better physicians than the Greeks because they treated the mind as well as the body.

PSYCHOSOMATIC INTERRELATIONS

THEIR THERAPEUTIC IMPLICATIONS IN GLAUCOMA

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During the past three decades many clinicians have become convinced that a patient is a unit consisting of body and mind, that there is a constant interrelation between the two components of this unit and that neither of them can be ill or well without the other being in a similar condition. The technical term for this body-mind unit has been coined psychosoma, and the interrelation between the two constituents is called psychosomatic interrelation. The idea is not entirely new, for twenty-four hundred years ago, on returning from military service, Socrates told his compatriots that the Thracians were medically ahead of the Greeks, that these barbarians knew that the body could not be cured without the mind and that the reason why the cure of many diseases was unknown to the physicians of Hellas was that they were ignorant of the whole. On another occasion, Socrates said: "Just as you ought not to attempt to cure eyes without head or head without body, so you should not treat body without soul." Hippocrates, who probably lived at about the same time, was of the same opinion: "In order to cure the human body, it is necessary to have a knowledge of the whole of things."

In modern times Ernst Fuchs has warned the ophthalmologic world that "we must remember we are treating patients, not eyes, and must therefore take into consideration all the physical and mental factors which may affect his outlook upon life. . . ." ¹

Although the systematic study of the psychic condition of patients, of their personalities, their mental conflicts and problems and their influence on the rest of the organism have been seriously pursued only for a few decades, the coming into being of a new era in medicine is already being witnessed: the psychosomatic era.

One may summarize some of the salient ideas concerning the psychosomatic interrelations published during the past thirty years as follows:

Read at the Seventy-Fifth Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 5, 1939.

1. Fuchs, E., quoted by Fasset, E. J.: *Some Thoughts on the Psychology of Refraction*, California & West. Med. **26**:53-54, 1927.

The relation between psyche and soma acts both ways: (a) Mental conflicts, emotional upsets, whether conscious or deeply buried in the unconscious, may produce a disturbance of physiologic processes and even cause reversible or irreversible organic lesions. (b) There is scarcely an organic disease which does not affect the psychic equilibrium or does not interfere with the normal pattern of a patient's personality.

During the first third of this century, the literature on psychosomatic interrelations became so extensive that the necessity arose of collecting in one volume the most important contributions on the subject. Dunbar² has published in one volume an excellent survey of 2,358 papers which have appeared between 1910 and 1933. (The papers on psychoanalysis are not included.) These papers concern every specialty of medicine. Ophthalmology is represented by a small number of contributions. The subject of glaucoma is treated in 8 papers, and only 5 of these have considered the subject seriously (if not exhaustively).

This paper is submitted partly for the reason that many leading medical men and several institutions in this country have considered the principle of psychosomatic interrelations of sufficient importance to introduce it into their routine methods of examination and mainly because the rank and file of ophthalmologists have ignored this important trend in medicine. As the material on this subject was somewhat lengthy and involved, it was divided in two parts. The first part, which was presented in a paper entitled "The Role of States of Anxiety in the Pathogenesis of Glaucoma,"³ included reports of a number of cases of glaucoma in which the ocular tension increased under the influence of states of anxiety. In this paper are discussed the principles of psychosomatic interrelations and the anatomic and physiologic substratum of emotions. It points out that the cerebral cortex, the thalamus and the hypothalamus (which contain the highest centers of the autonomic nervous system) together with the pituitary body and the adrenals form one unit within which the extremely complex phenomenon of emotions is developed and from which, by the aid of nerve pathways and hormones, the emotions are transmitted throughout the entire organism. The final chapter of the paper discusses the relation between the autonomic nervous system and the eyes and the influence excited by states of anxiety on the course of glaucoma.

2. Dunbar, H. F.: *Emotions and Bodily Changes*, ed. 2, New York, Columbia University Press, 1939.

3. Schoenberg, M. J.: *Role of States of Anxiety in the Pathogenesis of Glaucoma*, *Arch. Ophth.*, this issue, p. 76.

IMPORTANCE OF DIAGNOSIS FOR THE TREATMENT OF GLAUCOMA

If there is some truth in the contention expressed and argued in this previous paper,³ that the eyes of some patients with glaucoma react to states of anxiety by a rise of ocular tension, it follows that in addition to general medical and local treatment of the eyes, the patient's ego and his psychic difficulties and problems have to be treated as well. But, since a rational treatment cannot be instituted without a correct diagnosis, a few questions concerning the diagnosis of the emotional status of the patient need to be considered first.

Is the patient suffering from a psychic disturbance? What are the pattern and the nature of the disturbance? What is its underlying cause? Does the psychic disturbance play the role of a precipitating cause of glaucoma?

The physician who is supposed to clear up these diagnostic problems has to possess a certain type of personality and a good deal of knowledge and training regarding such matters.⁴ At the same time, the cooperation of the patient is essential. In a way, the picture of mental conflicts, repression, frustration, worry, excitement and distress is to be drawn by the patient himself under the cautious and discreet guidance of his physician. Once this is accomplished, the therapeutic implications are more plainly visible, both to the patient and to his physician. The fact of throwing light on the psychic situation has in many cases a therapeutic action.

REPORT OF A FEW CASES ILLUSTRATING THE THESIS OF THIS PAPER

CASE 1⁵—L. T. had in the course of time worked out his own psychic salvation almost completely. He found that states of anxiety due to financial worries, domestic squabbles, labor troubles and the fear of fire or explosions in his place of business were almost invariably accompanied by the perception of rainbow rings with his right eye. A change of environment, a short vacation in Florida or a week-end spent at his country home were sufficient to bring about a more quiet state of mind and a complete absence of visual complaints. After this situation had been clarified, the patient understood that the way of preventing attacks of ocular hypertension and deterioration of vision was by avoiding or eliminating mental upsets and by curbing overambitious business ventures, which are usually linked with increased responsibilities and worries. At the regular monthly visits at the office, the handling of this case consisted of routine examination of the patient's eyes and questioning him about his general health

4. Valuable information can be obtained by studying: (a) Schilder, P.: *Psychotherapy*, New York, W. W. Norton & Company, Inc., 1938. (b) Appel, K. E., and Strecker, E. A.: *Practical Examination of Personality and Behavior Disorders*, New York, The Macmillan Company, 1936. (c) Mohr, F.: *Psychophysische Behandlungsmethoden*, Leipzig, S. Hirzel, 1925.

5. The complete history of this case and that of the following one is recorded in a previous paper.³

(with which he was always satisfied) and about the condition of his eyes. At the same time a gently conducted, discreet survey of his attitude toward problems concerning his family, business and any other matters that might affect his psychic balance was made. In the course of time, a change in the patient's style and conception of life became evident. He acquired a certain poise and a desire of subduing overambition. He attributed the absence of attacks in the eye to the change in his point of view and more calm behavior.

CASE 2.—L. D., an inventor 63 years of age, called at my office on account of ocular trouble of several months' duration. He had subacute glaucoma in the right eye (tension, 65 mm. [Schiötz]) and simple glaucoma in the left eye (tension, 13.5 mm.). Hourly instillations of pilocarpine hydrochloride and applications of ice reduced the tension in the right eye to 17 mm. within twenty-four hours.

During the two years while under observation, the patient had a number of similar attacks at variable intervals. In the course of several months, through cautious questioning, the patient cleared up a good deal of the problem which puzzled him and me: Why did the attacks appear at certain times and not at others? He was instructed to keep a record of the events which preceded the onset of his attacks. Gradually he found that mental upsets, especially anxiety, were almost always followed by symptoms and signs of ocular hypertension. A long and stormy conference with the sponsors of his inventions during the night of Sept. 23, 1937 brought about a tension of 60 mm. (Schiötz) in his right eye. (This receded to 16 mm. after a two hour treatment with acetylbetamethylcholine chloride [mecholy] and prostigmine—a reversible situation.) "Hard thinking," calculating and designing for hours used to make him more excitable, and under such circumstances the right eye recorded this condition by a feeling of discomfort, foggy vision and perception of rainbows. Being an intelligent man, a mathematician trained to think logically, the patient soon began to realize that repeated attacks were injurious to the eye and damaging to vision and that the way to prevent the occurrence of such attacks was "to take it easy." Since he began to abandon the feeling of hastiness, the exaggerated eagerness and the long hours of steady work, his attacks became less frequent and less intense.

One day the patient brought along another interesting proof as to the relation of the condition of the eyes to that of his mind. He stated that ever since he lost his house (by not paying the mortgage on it) he felt much better. Both the mind as well as his eyes felt greatly relieved. The impending loss of his home had been the source of great concern for several years.

These 2 cases, selected from among several others, illustrate how mental upsets may have an influence on the eyes of patients with glaucoma and how psychodiagnosis established with the aid of the patient may have a therapeutic effect. However, no one would yet be justified to state that psychic upsets are always the precipitating cause or the most important cause of a rise of intraocular pressure. The psychosomatic interrelation in glaucoma is a problem which will need a long and careful investigation, and ophthalmologists are the most suitable persons to handle this problem. No doubt ordinary procedures, like direct questioning of the patient and repeated cautious survey of psychic difficulties, give at times unsatisfactory results. In such cases psychotherapists and psychoanalysts are more likely to succeed. But their

interpretations and conclusions are sometimes liable to be as erroneous as those of other clinicians, active in various fields of medicine. The position of some professional psychotherapists who are inclined to focus their attention only or principally on the psyche may be as narrow as that of clinicians, who think only in terms of organic diseases. The former act as if the patient is all mind; the latter, as if the patient consists only of soma.

The following 2 cases may illustrate how easy it may be to draw faulty conclusions.

CASES 3 and 4.—Two sisters, Reva and Anna, are considered together because of their remarkable disparate life history and because of the unusual opportunity offered us to realize the predominant influence of heredity in some cases of glaucoma.

Reva came under my observation one and a half years before the writing of this report, at the age of 57. She gave a history that for the previous eight months she had recurrent attacks of blindness. A diagnosis of simple glaucoma in each eye was made.

She had always been a placid person. She thought, talked and did everything slowly. Her education was of the lowest, and her intellectual requirements were almost equal to zero. She did not experience any difficulties in getting adjusted to married life. Besides taking care of her children and husband, there existed no other problems for her. She had not suffered from acute or prolonged states of anxiety since she came to this country, some thirty years ago. However, before she reached the age of 20 she went through four major emotional shocks. Within a few years she lost one brother and two sisters. Shortly after this she lived through the horrors of a pogrom, revolution and war. While the pogrom was taking place she was hiding in the attic of her house, and within three hours, so she related, the left side of her scalp became totally gray. In spite of all these violent states of anxiety which the patient had to live through, no ocular symptoms appeared. They did, however, appear more than thirty years later, when a simple, apparently benign glaucoma developed.

Anna, the sister, who was three years older, did not go through emotional cataclysms. She had left the old country at an earlier date. Her temperament was as placid as her sister's. Her psychic background presented no history of unusually intense excitement.

She came under my observation twelve years before the writing of this report, at the age of 51. A diagnosis was made of diabetes and acute glaucoma in the right eye and simple glaucoma in the left eye.

The right eye was soon operated on; the left eye behaved well for four years, but then acute glaucoma developed, which was treated surgically.

The subsequent history is not of interest here. The only question relevant to the thesis of this paper is: How does it happen that Reva, who went through some of the worst psychic experiences imaginable, had glaucoma more than thirty years later, and then under the form of an easily controllable simple glaucoma, while Anna, who had not lived through any harrowing experiences, had acute glaucoma six years earlier than her sister Reva?

Apparently, the predisposition to glaucoma inherited by both sisters was the paramount factor.

If by some chance only Reva would have been the patient under observation, one might have been inclined to believe that the serious emotional traumas which had been stored by unfortunate circumstances in the unconscious mind for some reason came to life thirty years later to precipitate the appearance of the ocular condition. Erroneous conclusions of this kind are likely to be met occasionally in psychoanalytic literature. Yet no one can deny the value of psychoanalysis even in its present form. The one thing that has to be guarded against is accepting blindly any one's conclusions and interpretations. The way of avoiding such errors is at least to familiarize oneself with the fundamentals of psychoanalysis and psychotherapy and, above all, to become interested in the personality problems of one's patients.

Schilder^{4a} enumerated a number of methods employed by those trained in the technic of psychotherapy: (1) discussion with the patient about his psychic problems, (2) advice, (3) persuasion, (4) appeal to will power, (5) discussion of the past, (6) cathartic hypnosis, (7) free association (psychoanalysis), (8) dream interpretation (psychoanalysis), (9) interpretation of the mistakes of everyday life, (10) analysis of ideologies, (11) analysis of social adaptations, (12) suggestion, (13) relaxation and concentration, (14) change of surroundings and (15) occupational therapy.

The multitude of the methods is a warning that one should "not overestimate the chances of psychotherapy or underrate its difficulties" (Schilder). My own experience has been mainly the result of a keen interest in the psychic problems and difficulties of my patients with glaucoma. Of the various psychotherapeutic methods enumerated by Schilder, those which are linked to psychodiagnosis seem to be the most promising of success. The difficulties which are being gradually "discovered" by the patient himself are more likely to be solved than those pointed out by the physician alone. For this reason, advice, persuasion, appeal to will power, the physician's interpretation of everyday mistakes, suggestion and the other methods are less likely to succeed. The gradual, cautious and discreet inducement of the patient to "list" and discuss his psychic difficulties and a study of his dreams by a competent psychotherapist are a more certain way for his finding a remedy for his difficulties. Any one or any combination of the following remedies—a change of surroundings, elimination of sources of friction or reappraisal of these sources as to their real existence or seriousness, a readjustment to the environment, occupational therapy or an avocation—are likely to change the psychic situation for the better and to revert a somatic dysfunction if it is still in the reversible stage.

CONCLUSIONS

1. A somatic diagnosis without a survey of the patient's emotional life is an incomplete diagnosis.
2. Psychodiagnosis is essential to psychotherapy.
3. Psychotherapy is sometimes as essential for patients with glaucoma who are under the influence of states of anxiety as somatic therapy.

DISCUSSION

DR. J. W. CHARLES, St. Louis: After the Elliot trephine operation for glaucoma was demonstrated in 1913-1914 I saw the good Samaritan of her village, a woman who nursed the sick, laid out the dead and attended all the funerals. She came to me with absolute glaucoma and in great pain. I employed trephining. I asked the patient to see me once a month after that. The other eye was perfectly normal. She came in for a few months, and I advised her to stop her outside activities and especially to stop attending funerals. As she neglected to see me for a few years, I had the idea she did not entirely obey me. After five, or perhaps more, years she came in with acute glaucoma in the other eye. Her 42 year old son had broken his leg. In spite of his having a wife, the mother nursed him, and her anxiety expressed itself in an attack of acute glaucoma. Her blood pressure was then so high and her vision quite low that I did not dare operate at once. I prescribed spirits of glyceryl trinitrate and, as I remember, sodium nitrite; on either the next day or the second day her blood pressure was much lower, but her vision had decreased almost to perception of light in her good eye overnight. I trephined the eye in the office, and took the patient to the hospital in a cab. She recovered her vision, and I saw her some years afterward in fairly good condition, except that the eye had become a little soft. She had lost some of her vision, but the last time I saw her, ten years ago (that covers a history of fifteen years), it was still fairly good.

I think every physician should know something of his patients' background and that every ophthalmologist should know something about their mentality and their emotions. I believe that would be a great help in treating glaucoma.

DR. F. PARK LEWIS, Buffalo: I think that if one dares to criticize a branch of medicine which has advanced so definitely during the last two or three decades as ophthalmology has it would be to say that ophthalmologists have particularized too accurately. The direction of opinions has been localized more specifically on the eye than on the conditions on which ocular changes are frequently dependent. Some time ago I asked a group of ophthalmologists from different parts of the country to discuss the possible causes and the possible measures of relief of simple glaucoma. There were as many opinions as there were men present. I asked a similar group in London last year, in which there were representatives of eight different countries—men who are well known—to discuss the same question. I said: "Do you agree with me as to the importance of the knowledge as to the cause or causes of simple glaucoma?" They all agreed. I said: "Do you believe that the study of the origin of simple glaucoma would be worth

while?" They all agreed that nothing could be more worth while. We've said that a discovery of the conditions that would eliminate any of the causes of glaucoma would be priceless. It may be that Imre is correct when he said that glaucoma is not one disease but a group of diseases in which the main symptom is increased intraocular tension. If that is true—and I think ophthalmologists are growing to believe that it is true—then there is nothing more important than to study contingent conditions associated with intraocular pressure. That they are neurotic and psychic in certain cases everybody knows. There is scarcely an ophthalmologist who has not had experience with persons in whom emotions have resulted in acute attacks of glaucoma, but there may be other conditions. I have had under my observation for twenty-five years a man who has periodic attacks of increased intraocular tension—an observing professional man who knows the causes. He knows that they are due to excesses—excesses of food, excesses sexually, excesses in various things which disturb the nerve centers—and that by the avoidance of these and sometimes by the use of a little pilocarpine, he is able to overcome these attacks; thus the state of his left eye, in which the increase in tension occurred, has continued to be normal for the past quarter of a century. A few cases of this kind—they need not be common—demonstrate the possibility of such factors being at least one of the causes of increased ocular tension. I would not go so far as to suggest the possibility of pushing Dr. Schoenberg's thesis that the ophthalmologist should become a psychoanalyst, but he must cooperate more with his confrères in the general medical profession. He must find some of the contingent and contemporary conditions that occur with glaucoma and not confine his attention exclusively to local and operative measures.

DR. HANS BARKAN, San Francisco: Dr. Schoenberg has touched on one of the most difficult problems with which the ophthalmologist has to deal. I am anxious to read his paper. I am sure he is aware of one historical incident that he has not had time to bring out. It concerns one of the great ophthalmologists who had his first attack of glaucoma while in a state of anxiety and anger when attending the Dreyfuss trial in 1879 and gives striking historical evidence of the effects of emotion in starting such an attack. I was interested to hear Dr. Schoenberg speak of the relation between body and mind in these patients. Few ophthalmologists have the time to go into the question exhaustively, and all will agree there are a certain number of patients who have more body than mind. These are best left untreated, and if one has some clinical acumen one can decide that such persons are not worth bothering about as far as mind is concerned. However, one can have some influence on the minor number who have a mind. Here again there are two broad methods of approaching the question. I feel that the psychoanalytic approach to their problems by a professional in that branch is a poor way to treat the majority of these elderly persons. It stirs up impressions and starts them worrying. If one had in the office the tools with which to handle their problems, it would be the practical thing to try to do. I have been much interested in the question, because for some reason or other I have had an inordinate number of cases of glaucoma compared to other surgical conditions. I have found a rather practical way of solving the problem. As Dr. Schoenberg said, it

depends on the mind of the patient and how one gets along with him by talking to him, but the most successful practical way, for me at least, has been as follows: After the diagnosis is made, if the condition is operable it is not the time to start with the relation of the patient's mind to his glaucoma. I say to the patient: "It is my responsibility as far as the diagnosis and the operation are concerned. The minute you come into the hospital that eye does not belong to you—it is my eye. You have worried about it long enough; you have had pain in it long enough. I will return the eye when we are through operating." It depends on the patient and partly on the ophthalmologist as to whether he succeeds. He will succeed in some cases at least. Afterward, when the operation is concluded, one can go into the effect of the mind, perhaps along the lines which Dr. Schoenberg does. I am not sure. As far as the emotional aspect is concerned, it has some bearing in almost all cases. It was Cannon, in this country, who showed the influence of anxiety, fear, anger and similar emotions in his work on the gastrointestinal tract. It is known that there are glaucomatous attacks which are undoubtedly dependent on the degree of worrying that the patient does and which are coincident with the existence of deep grief.

It is a large field, and I shall be most interested to read the paper in full.

DR. F. H. VERHOEFF, Boston: I think that all ophthalmologists when they are first told anything about glaucoma are taught that emotions precipitate acute attacks; they are also taught that when treating a patient with acute glaucoma a miotic should be put in the other eye and that if the patient is operated on, whether the glaucoma is acute or not, the other eye should be treated with a miotic. As a matter of fact, I have more confidence in the miotic than I would have in turning Dr. Schoenberg loose on the patient or in trying to persuade the patient to be calm, because it is difficult for a person with acute glaucoma or a painful eye of any kind to be calm. I think pilocarpine or physostigmine will have a more calming influence on the other eye than anything one can tell the patient. I do not understand how Dr. Schoenberg finds the time to go into the mental qualifications and emotional conditions of these patients. I know my emotional condition would not permit me to do so. I think the only way out would be to have a board to train nurses in the psychology of these patients, and perhaps in that way there would not be as many persons with acute glaucoma consulting the ophthalmologists.

DR. CARL KOLLER, New York: I should like to contribute something to the knowledge of the interrelations of emotions and glaucomatous conditions. The first man to do this was Weber, who was an ophthalmologist in Odessa. He reported that every time he played cards he had an attack of glaucoma. That was more than fifty years ago. Javelle had a prodromal attack, and he said that he made the glaucoma worse by trying to cure it with cocaine, which he instilled himself. Javelle was not an ophthalmologist but an optical engineer, and that likely accounts for his not knowing that a mydriatic would be the wrong thing to use.

DR. EDWARD JACKSON, Denver: I think that a couple of experiences which I have had have directed my attention to this channel and are

a propos to the paper and the discussion. The first year that the United States went into the World War a buyer for a large drygoods store in Denver had an acute attack of glaucoma while in New York. She was referred to the younger Dr. Callan. He had already gone into service, so she went to Dr. Peter Callan, a former member of this society and a conservative and sound man in ophthalmology. He did an iridectomy on one eye and immediately began to use physostigmine in the eyes. The glaucoma was bilateral according to the history, and the patient was told that she had to have the other eye operated on. She did not want to remain in New York for the operation and subsequent treatment, so Dr. Callan wrote me a letter telling the condition of the eye, the tension of the eyeballs when he first saw her and the tension of the eyes when he last saw her, which was perhaps three weeks after the operation. He also told her to see me and have the second operation done. When she arrived in Denver I found the tension of the eye which had been operated on to be perfectly normal. The iridectomy was excellent, and I felt sure that the eye was saved. I told the patient that, and I also told her that when I first took the tension of the other eye it was distinctly lower than it had been when Dr. Callan last saw her, just before she left New York, and that I might try miotics, physostigmine at night and pilocarpine during the day for a little while, as it would be better for her to get into good condition before she had the second eye operated on. Within a couple of weeks there was a distinct lowering of the tension. Up to that time it was 30 to 40 mm. of mercury in the eye which had not been operated on. Within two weeks it was distinctly better, and I talked the situation over frankly with her and advised her to continue the miotics for a time longer, as it was possible that an operation could be avoided. She did and was very happy about the situation; the consequence was that within six months the use of pilocarpine was discontinued entirely and both of her eyes were well. After the close of the World War she had an opportunity to buy silks in China and Japan. When she went out there she took a bottle of solution of physostigmine and a prescription for it along with her as well as a brief account of her case if it were needed. She never had any further signs of glaucoma. She came back and twenty years later has had no return of the symptoms.

The other case was that of a woman who came to Colorado with her husband, who had tuberculosis. At that time outdoor treatment was beginning to be used entirely. She and her husband lived in a tent, and in the autumn he grew worse and died. For three days before his death she was with him continuously, exposed to the cold, and was constantly interested in the outcome of his illness. After his death, she came to me with acute glaucoma in both eyes. I thought strongly at that time that it was inadvisable to operate if the operation could be put off. I talked to her about it frankly and told her that there was a chance by the use of miotics of escaping an operation. She seemed to recover rather remarkably, considering the great strain and anxiety she had been under, and it was only a few days before I could encourage her with the hope that the operation would not be necessary on either of her eyes. They were both improving, and within a month she stopped the use of the miotics entirely; during the three years she lived in Denver she never had another attack of glaucoma, not even a mild one.

One other disease which ought to be considered in this connection is migraine. I can cite a case of migraine in which there was less than a diopter of error of hyperopia in either meridian of each eye. The patient, a girl of 15, who was rather poorly prepared for the grade in school in which she was, had migraine several times in a month and sometimes as often as twice a week. She put on weak glasses. From that time she did not have any attacks of migraine. In about three weeks she thought she had recovered from her trouble. She left off her glasses for one day and had an attack. Perhaps three months after that she broke her glasses and was without them for one day. That day she had another attack. She has since been in good health. She studied nursing, married and has three healthy children, and for the last twelve years she has not worn glasses at all.

MR. H. M. TRAQUAIR, Edinburgh, Scotland: I feel greatly honored by being asked to discuss this paper, and I should like to say that I have long been of the opinion that glaucoma is a disease of the patient and not of the eye; that is to say, that glaucoma is, like the majority of ocular diseases, a manifestation of a general condition. If one does not know exactly what that general condition is, that does not modify the truth of the thesis, or hypothesis, I should say. I voiced this opinion four years ago at a meeting of the British Medical Association, and the idea was received with a great deal of opposition. I am interested to hear that opinion is changing to the idea that glaucoma is a disease first of all of the individual and secondly of his eye. That brings up the question of therapeutics. I need not quote examples of the numerous cases all have experienced in which an emotional upset has produced an attack of glaucoma. Every ophthalmologist has had experience with such cases. With regard to therapeutics, it is, of course, essential that the patient's environment should be examined, but I rather doubt that mental treatment would be, in the present state of the ophthalmologist's ability to conduct it, as efficient as the use of pilocarpine or physostigmine. I think I agree with Dr. Verhoeff on that point; as an oculist in practice I should prefer to administer the physostigmine and to leave the mental treatment—not that I wish to disparage the idea at all—for a less urgent occasion. I agree thoroughly with the view that glaucoma is determined to a large extent by the mental condition and that it is a disease of the patient, but when one considers hereditary glaucoma it is difficult to see how one can rely to any great extent on psychic treatment of any kind in these cases, though I think it should not be omitted.

DR. MARK J. SCHOENBERG, New York: The cases reported by Dr. Charles and Dr. Jackson and the remarks of Dr. Park Lewis and Dr. Verhoeff confirm my experience regarding the close relation between acute emotional upsets and the outbreak of an acute glaucomatous crisis.

It seems to me that through some misunderstanding Mr. Traquair, Dr. Verhoeff and others have the impression that I am advocating the study of psychosomatic relation and psychotherapy for patients with acute glaucoma. The study of a patient's psychosomatic relation takes weeks and months; psychotherapy cannot be applied without a careful

psychodiagnosis. Acute glaucoma is a serious crisis that cannot wait. Therefore, psychotherapy is not applicable to patients with acute glaucoma. This does not, however, mean that once the emergency of the acute attack has been taken care of, there is no need for an investigation of the patient's psychic problems and difficulties. His other eye may yet be in the early stage of glaucoma, and psychic upsets may act in the future as a precipitating factor of ocular hypertensive crises. Even in hereditary glaucoma, mentioned by Mr. Traquair, one may not go wrong by investigating the psychic background of the patient. The information obtained along these lines may help in preventing an acute glaucomatous crisis.

Dr. Park Lewis' belief that "excesses of any kind" (such as those pertaining to the sexual act, food and work) may precipitate an increase of ocular tension is shared by most ophthalmologists; but the subject of my paper is limited to the role of states of anxiety in glaucoma and to therapeutic implications.

Dr. Verhoeff rejects the idea of investigating the psychosomatic relation in cases of glaucoma mainly on two grounds: (a) He has no time or inclination for this sort of work. (b) He prefers to rely on miotics rather than on telling the patient to be calm. The lack of time to make a complete diagnosis cannot be considered as a valid reason for rejecting an additional method of examination. Surely the patient has a right to demand that a physician devote all the time necessary to make a complete diagnosis. As far as Dr. Verhoeff's greater faith in miotics than in psychotherapy is concerned, I wish to call his attention to the fact that my paper does not advocate giving up the use of miotics—surely not at the present stage of ignorance about glaucoma.

The feeling was almost unanimously expressed that pilocarpine and physostigmine salicylate are more efficient than "telling the patient to be calm." I must repeat that my paper is concerned with psychosomatic relations in glaucoma and their therapeutic implications and not with pharmacologic or surgical treatment. Furthermore, psychotherapy does not consist in telling the patient to be calm. The sooner and the more thoroughly one becomes informed on the subject, the better off one's patients will be.

Dr. Verhoeff's suggestion to place the psychosomatic study in the hands of nurses is not a very happy one. It is my belief that only physicians should handle such problems. I am certain that the quality of their work would not be enhanced by relegating some of it to nurses.

Dr. Barkan also seems to complain about the lack of time for going into the detailed study of psychic conflicts and upsets. He also thinks that elderly persons should be spared an investigation of this kind since only a minor number among this group "has a mind." Few physicians will agree with this assumption, which, I believe, is not supported by facts. Dr. Koller's patient is similar to one of my own, who almost invariably had a transient glaucomatous crisis whenever he played cards.

No one needs to become upset by the fear that psychoanalysts may claim glaucoma as belonging to their field of research. Psychoanalysis

is a remarkable contribution to the sum total of human knowledge, especially to abnormal psychology. But there is still a long, long way to go before psychoanalysis can be considered as a more or less finished product. Ophthalmologists have always availed themselves of the progress made by various branches of science, and if psychoanalysis will prove itself useful, it will surely be accepted. But, all this does not mean that I advocate that ophthalmologists have to practice psychoanalysis. They could not do it even if they wished.

I am sorry that Dr. Bedell did not discuss my paper, and I am at a loss to know what was the reason for his upset state of mind.

THE LELAND REFRACTOR

A METHOD FOR REFRACTION UNDER BINOCULAR CONDITIONS

STEFAN VAN WIEN, M.D.

CHICAGO

The Leland refractor, designed by George H. Leland, an electrical engineer, of Dayton, Ohio, is an instrument for use in refraction under binocular condition.

The visual targets are placed opposite the patient at a distance of 6 meters, or they are placed beside the patient and are viewed in a mirror at a distance of 3 meters. One of the targets (fig. 1, *A*) is a slit, 4.8 mm. ($\frac{3}{16}$ inches) in width and 8.5 cm. ($3\frac{3}{8}$ inches) in length, which can be rotated to any desired meridian and is evenly illuminated by diffused red light. The large dial (fig. 1, *C*) makes it possible to read easily single degrees of the axis of astigmatism. A second target (fig. 1, *B*) consists of two slits at right angles, having the same measurements and forming a cross. The same unit also provides a muscle light (fig. 1, *D*) and a visual acuity chart (fig. 1, *E*).

This method is employed to its best advantage with a standard phoropter, though a phoropter is not essential. The extension rod of the phoropter is provided with a muscle chart (fig. 1, *F*) to measure muscle imbalances for close range.

Each of the two targets is diffusely illuminated by the reflected light of a silver screen, which in its turn is illuminated by two bulbs that flash alternately. One set of bulbs is controlled by a common rheostat (fig. 1, *G*) and is adjusted to rather a low intensity, just enough to furnish a stimulus for fixation. The other set is adjusted by another rheostat (fig. 1, *H*) to rather a brilliant illumination. There are, therefore, two periods, a dim and a bright one. The ratio of the duration of the dim period to the bright one is 2:1. In front of each of the four bulbs is a polaroid film, the axis of which for the dim bulbs is adjusted vertically and for the bright bulbs horizontally. Another polaroid filter (fig. 1, *I*) with its axis vertical is attached to the phoropter and can be turned to cover either eye. It is evident that during the dim period, when the axes of the polarizing and the analyzing filter coincide, the target is seen by each eye in the same intensity. One would think, however, that during the bright period the target should not be seen at

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all by the eye with the analyzing filter, but this is not the case. Red light is not completely polarized by polaroid film, and it is partly depolarized when reflected from the silver screen. During the examination the patient is under the impression of seeing the target, bright and dim, flash up before both eyes. In reality, during the bright period only the eye under investigation perceives a bright image of the target, whereas the eye with the polaroid filter perceives only the faintest impression, not strong enough to interfere with the testing of the other eye but still sufficiently strong to stimulate it for fixation. The incom-

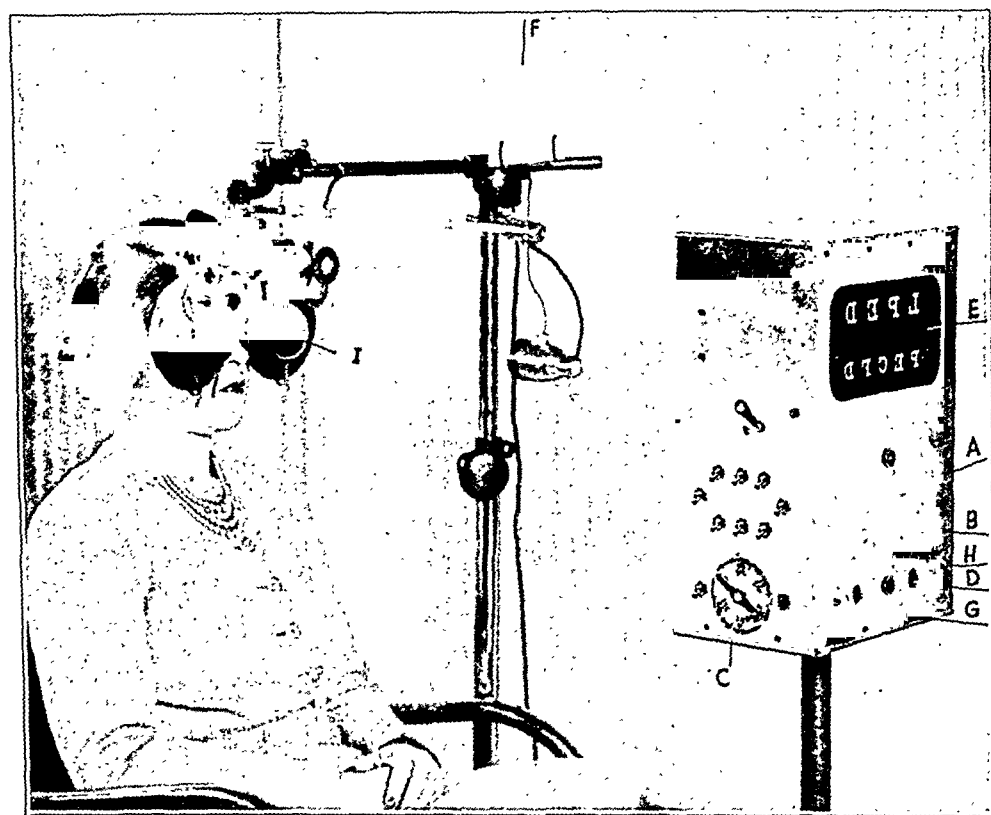


Fig. 1.—The Leland refractor. *A* indicates the single line target; *B*, the cross target; *C*, the astigmatic dial; *D*, the muscle light; *E*, the visual acuity chart; *F*, the muscle chart; *G*, the rheostat for the dim period; *H*, the rheostat for the bright period, and *I*, the analyzer filter.

pleteness of polarization guarantees a constant fixation of both eyes during the whole test.

The procedure for subjective refraction with the apparatus is as follows: If nothing is known about the refraction of the eyes to be examined, astigmatism with the rule is assumed. The single line target is set at 180 degrees. The polaroid analyzer is placed in front of the eye which is not being subjected to refraction. During the dim period the spherical lenses in the phoropter are changed—either in one-eighth or one-fourth steps—till the patient reports that the line appears clearest

and narrowest. The next step is to determine the correct axis of astigmatism. The target is now kept constantly bright, and the spherical correction is slightly altered toward the minus side. Next the target is rotated slowly through 180 degrees, and the patient is asked to indicate at what meridian it appears sharpest and thinnest. The latter then determines the axis of the negative principal meridian. The flashing device is turned on again, and the proper spherical correction for this meridian is determined. The correction for the positive principal meridian is then found by rotating the target 90 degrees and by repeating the maneuver. The test is continued by turning from the single line to the crossed lines. If the procedure so far is correct, the two crossed bars should appear identical in width if the spherocylindric correction which was found is inserted in the phoropter battery. Should there be a slight difference in the width of the two bars, an increase or decrease of the cylindric correction should compensate for it. It is of relatively frequent occurrence that the horizontal line cannot be seen as thinly as the vertical, a phenomenon long ago observed by Helmholtz in similar instances. Anyhow, the patient can determine when the width of the two crossed bars is as much alike as possible. The correction thus found is left in the lens battery, the polaroid filter is switched to this eye and the identical procedure is repeated for the other eye. In order to get a complete spherical balance of both eyes, one now goes back to the eye examined first and rotates the crossed lines to the 90—180 degree position, for the cylindric correction is now assumed to be right and one should not have to make any new adjustment in the cylinders. Some change in sphere, however, might render the target clearer. The same test is repeated for the other eye. Only at this stage is one taking the visual acuity for each eye individually. By the help of the crossed cylinder, slight changes in the amount of astigmatism or its axis may improve the visual acuity, but no other changes should be made in the spherical correction. The next step is to check the muscle balance and the fusion amplitude for distance. With patients of the presbyopic age, the reading addition is found next, and then the muscle balance for close range, either with or without the reading addition, as the patient may require, is determined. Finally, the distance correction is adjusted once more, binocularly with the visual acuity chart. If there was a difference between the axis binocularly as tested with the target line and monocularly as tested with the visual acuity chart, one also determines which axis is accepted when the test is done binocularly with the visual acuity chart.

The principal difference between this new method and any other method is that with the former one examines the eyes under binocular conditions. This is of importance for the proper spherical balance of both eyes and might play an important part in changes of the axes in

cases in which a high cylinder is required, as I shall demonstrate later. As far as the spherical balance of both eyes is concerned, it should be noted, as proved by Hess¹ and Neumann, that one is unable to compensate for anisometropia of even 0.12 diopter by unequal accommodation, because both ciliary muscles act equally and simultaneously. While anisometropic eyes or eyes that are made anisometropic by faulty correction still might get equally good visual acuity, it is surely preferable to give an ideally balanced correction which places the posterior focal point for light of the same wavelength on corresponding layers of the retinas. To insure that both eyes are in the same state of accommodation during the period of examination, the test has to be done under binocular conditions.

The proper spherical balance between the two eyes is also important from another point of view. For distant vision there must be parallelism of the two visual axes during the act of binocular vision, for otherwise the subject would suffer from diplopia. In other words, in case of exophoria there must be convergence in order to avoid diplopia and in case of esophoria there must be divergence. I want to consider for a moment the correlation between convergence and accommodation. In order to simplify the digits, I shall assume that the interpupillary distance is 60 mm., or 6 cm. A prism that has the strength of 1 prism diopter deviates a beam of light 1 cm. at a distance of 1 meter on a tangent scale. The convergence for 1 meter would be 6 prism diopters; for 0.33 meter the convergence would be three times as much, or 18 prism diopters, and for 2 meters it would be only one-half as much, or 3 prism diopters, and so on. At the same time the accommodation would be 1 diopter for 1 meter, 3 diopters for 0.33 meter, 0.5 diopter for 2 meters, and so on. In other words, in case of a 60 mm. interpupillary distance, the convergence accommodation ratio would be 6:1. This means, of course, that each prism diopter of exophoria requires 0.166 diopter in accommodation, and each prism diopter of esophoria, the relaxation of 0.166 diopter in accommodation. To give an example: If a patient has an exophoria of 3 prism diopters at infinity, he has to converge these 3 prism diopters in order to avoid diplopia, and at the same time he will accommodate 0.5 diopter. Then, of course, his punctum remotum is no longer at infinity, but at a distance of 2 meters, and everything beyond this distance will look blurred. In order to correct this condition, one simply adds — 0.5 diopter to the distance correction, and though the convergence-accommodation ratio is preserved, the punctum remotum is brought again to infinity. There are obvious limitations to this kind of correction. In the first place, in case of

1. Hess, C.: Arbeiten aus dem Gebiete der Accommodationslehre, Arch. f. Ophth. (pt. 2) 42:80, 1896.

esophoria one cannot relax accommodation above a certain natural limit by adding plus sphere; i. e., the accommodation is already completely relaxed. Furthermore, in case of marked exophoria one cannot add too great a burden to the accommodation for distance.

In every instance of muscle imbalance it should be possible to figure out how this imbalance can be corrected by spherical changes only. Actually, the problem is not so simple. Accommodation is not the only factor responsible for convergence. The other factor is the desire for fusion, and one therefore speaks of total convergence as the sum of accommodative and fusional convergence. This sum might be reached by an indefinite number of ways, as illustrated in the following contrasting cases: One patient has orthophoria for a distance of 6 meters and at 0.33 meters, the other has 1 prism diopter of exophoria for a distance of 6 meters and 18 prism diopters of exophoria for 0.33 meter. Both persons are actually orthophoric at infinity. In the first instance the total convergence is accommodative and in the second case the total convergence is fusional. A third factor to be considered is the so-called "Pannum's areas," which means that for actual single vision the retinal images do not have to be exactly at corresponding retinal areas, a psychophysiologic phenomenon that goes entirely beyond the realm of mathematics. The Leland refractor automatically adjusts for these involved considerations.

The majority of patients will, of course, be able to rearrange the convergence-accommodation relation, but still there are a number who have difficulty. The latter should be benefited by a test with the Leland refractor. Patients of this type wander from one office to the other, never feeling quite comfortable.

The other advantage of this new instrument lies in the determination of the axis of astigmatism under binocular conditions. It is assumed that the negative torsional effect of the superior rectus and superior oblique muscles is neutralized by the equally strong positive effect of the inferior rectus and inferior oblique muscles. If in some cases, particularly in those of high astigmatism, this does not occur, a binocular determination of the axis of astigmatism should be of great advantage. Primarily, I have in mind cases of cyclophoria. While tests for vertical or horizontal muscle imbalances are more or less a routine procedure, one only rarely tests for cyclophoria or cyclotropia. The investigations of Stevens and of Verhoeff² reveal that this anomaly does not occur so rarely as is generally believed. Verhoeff was of the opinion that only fusional compensation takes place in these cases. Ames,³ on the other

2. Verhoeff, F. H.: Description of a Reflecting Phorometer, *Am. J. Physiol. Optics* 7:39 (Jan.) 1926.

3. Ames, A., Jr.: Cyclophoria, *Am. J. Physiol. Optics* 7:3 (Jan.) 1926.

hand, definitely expressed the belief that there is a torsional compensation in cases of cyclophoria, though he also stated that fusion is partly responsible for the compensation. Hence it is desirable, especially in cases in which a high cylindric correction is needed, to have some means of determining axes under actual working conditions.

Clinical examinations were conducted in the ophthalmologic department of Northwestern University and in the outpatient department of Mount Sinai Hospital. At Northwestern University I first did refraction under cycloplegia, after retinoscopic examination was done by the refractionist. During cycloplegia, of course, I disregarded the muscle tests and attempted only to get a basis to test the reliability of this method.

Without discussing individual cases, I wish to state that the results practically were identical. There was, of course, some difference between the results of the postcycloplegic test and the results obtained by the Leland refractor, but that was what I expected. In practically all instances the results obtained by this instrument showed either orthophoria for distance or at least less muscle imbalance than when the ordinary postcycloplegic test was performed. At Mount Sinai Hospital, where I did the retinoscopic examinations, I used the Leland refractor as a rule only for postcycloplegic tests. As far as cooperation on the part of the patient was concerned, I had to discontinue the test or disregard the results five times among approximately 100 clinic patients. The patients usually showed a great deal of enthusiasm and interest for this new type of examination. There were even several children 10 years of age among my patients.

It is possible to complete an examination within twenty to thirty minutes if one masters the new technic and knows the retinoscopic findings. For the patient the new type of examination is by no means tiresome or strenuous. As far as office routine is concerned, it would be impracticable to use this method in every case, but I believe that it would be of great advantage to use it in indicated cases and whenever time permits.

In considering possible objections to the method, the following problems come to mind. If the eye which is not being investigated does not recognize any change in the appearance of the target, it might suppress altogether and assume its position of anatomic rest. This is actually not the case. If there is no marked muscle imbalance, one creates an artificial esophoria by placing a strong prism of about 10 prism diopters, base out, in front of the eye not under investigation. If the binocular vision were interrupted during the bright period, it would be noticed that during the dim period the eye would turn in. But this does not happen. The image which belongs to the eye with the polaroid film in front of it is always fused with, or at least superposed on, the bright

image. There are, however, some cases in which muscular imbalance cannot be corrected by spherical changes. In such cases the two images are fused by means of the rotary prism, and here one has at the same time the exact amount of prism that should be incorporated into the final prescription.

The red color was not chosen unintentionally but by trial was found to give the best results. This is perhaps contrary to what might have been expected. Without going extensively into the matter of chromatic aberration, one knows that the chromatic aberration of the human eye amounts to about 1.75 diopters, and it is generally assumed that the eye unconsciously chooses the brightest part of the spectrum for its focus, i. e., the yellow of about 5,900 angstrom units.

So far I have been unable to find any references that red is less likely to stimulate accommodation than other monochromatic colors. However, this seems to be the fact. Ordinarily I use a plain red gelatin

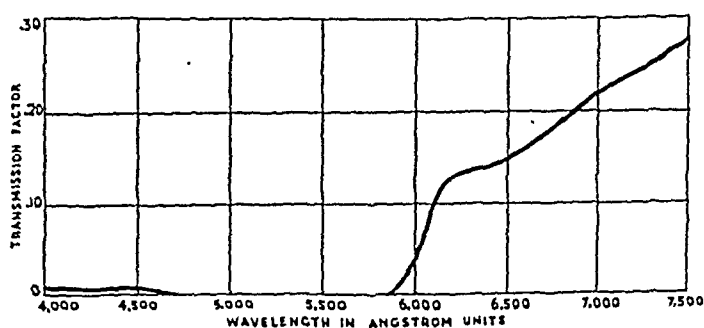


Fig. 2.—Spectrophotometric transmission curve of red filters.

filter which is not purely monochromatic but has a spectrophotometric transmission curve, as depicted in figure 2. There are no differences in the result if a Wratten filter no. 61 is used which is transparent only for red light of between 7,000 and 7,200 angstrom units.

Since there is no Wratten filter with a transparency only for yellow of 5,900 angstrom units, I used the one that comes closest to it, namely, a green one (no. 29) which is transparent for light of between 5,600 and 5,800 angstrom units. When the eye is tested by the Leland refractor and is under the influence of a cycloplegic, one gets a definite result by utilizing the green filter. The findings, however, do not coincide with those obtained by retinoscopic examination. They were about a whole diopter more toward the minus side for 6 selected patients whom I examined. In my clinical investigations I used the Leland refractor with the red filter during cycloplegia for about one third of the patients. For all the result was practically the same as that found by retinoscopic examination. I also compared the results obtained with velonoscopic examination. With this method, if one looks at a distant source of light

and moves an object in front of the eye—for instance, a wire—close to the pupil, one observes the spatial projection of the shadow that the object casts on the retina. The movements are identical with those observed on retinoscopic examination with a concave mirror, in other words, “with movements” in a myopic and “against movements” in a hyperopic eye. For a trained observer this method is accurate to 0.12 diopter, with a possible limit of 0.06 diopter. If the neutral point is reached, no movement is observed. The results obtained by velonoscopic examination with the eyes under cycloplegia are identical with those obtained with the Leland refractor for corresponding filters, and in case of the red filter, with those obtained by retinoscopic examination.

If the subject should accommodate for red, he would see retinal images of different size, but absolutely clearcut. The difference in size, however, could not be noticed. If, for instance, he should try to overcome by accommodation the effect of a — 3.00 diopter lens held over the distance correction, the difference would be only 1 per cent. The only possible explanation, then, for the difference in size that is noticed with this arrangement is that the subject does not accommodate and notices blurred circles. Helmholtz⁴ developed a method to calculate the size of blurred circles. With my arrangement, the size of the blurred circles, as caused by ametropia of only 0.12 diopter, would be between 10 and 15 per cent of the width of the target, according to the size of the pupil. It is evident that it should not cause any difficulty, even for an untrained observer, to appreciate that difference.

SUMMARY

An instrument is presented which by the use of polarized light enables one to conduct refraction under binocular conditions.

The advantages are not only a sensitive test for the axis and amount of astigmatism but an instrument which permits a balancing of the spherical correction as well as adjustment of the axis of the cylinders in binocular vision.

In cases of muscular imbalance the instrument indicates the spherical changes required or, if prisms are indicated, the proper amount.

104 South Michigan Boulevard.

4. Helmholtz, H.: Helmholtz's Treatise on Physiological Optics, translated from the third German edition, edited by J. P. C. Southall, Ithaca, N. Y., The Optical Society of America, 1924, vol. 1, p. 130.

OCULAR LEPROSY IN THE UNITED STATES

A STUDY OF 350 CASES

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ST. PAUL

The literature of ocular leprosy in the United States during the past few decades has for the most part consisted of scattered case reports. Thus, Yudkin,¹ Lane,² Levitt,³ Horton,⁴ Rogers,⁵ Crebbin⁶ and Pfingst⁷ have contributed observations. Pinkerton⁸ and Van Poole⁹ have presented valuable findings on lepers from the Hawaiian Islands. This scarcity of literature can be attributed to the fact that few ophthalmologists in this country have studied leprosy, owing to its relative infrequency (about 1,000 estimated cases in the United States). Also, ocular lesions of leprosy have proved so resistant to treatment that this field has been termed by some a hopeless branch of ophthalmology. Blindness is the most dreaded complication of persons afflicted with leprosy, and observations at the national leper colony readily convince one of this fact and make one realize the problem confronting officials in countries where the disease is so much more prevalent.

At the request of the United States Public Health Service, I recently made an ophthalmic examination and study of patients in the National Leprosarium at Carville, La. Three hundred and fifty of the 370 patients in the hospital were examined.

This examination included testing of vision, external examination of the lids, brows, conjunctiva, lacrimal sac and extraocular muscles; external and slit lamp examination of the cornea and iris; tonometric measurements; visualization of the lens, and, when such was possible,

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1. Yudkin, A. M.: *Am. J. Ophth.* **1**:303 (May) 1918.
2. Lane, J. E.: *New York M. J.* **104**:1244 (Dec. 23) 1916.
3. Levitt, M.: *New York M. J.* **116**:376 (Oct. 4) 1922.
4. Horton, J. J.: *Am. J. Ophth.* **8**:382 (May) 1925.
5. Rogers, R. M.: *Am. J. Ophth.* **10**:503 (July) 1927.
6. Crebbin, A. R.: *Am. J. Ophth.* **12**:384 (May) 1929.
7. Pfingst, A. O.: *Am. J. Ophth.* **9**:195 (March) 1926.
8. Pinkerton, F. J.: *Arch. Ophth.* **56**:42 (Jan.) 1927.
9. Van Poole, G. M.: *Tr. Am. Ophth. Soc.* **32**:596, 1934.

ophthalmoscopic examination of all fundi with the hand and binocular ophthalmoscope. The race, age, sex, duration and type of disease were also recorded. Twenty-eight enucleated eyes were sectioned and studied. Various types of local and systemic treatments were tried and the results watched for several months. Thirty operations were performed. Photographs, photomicrographs and lantern slides were made of the principal ocular lesions.



Fig. 1.—Cutaneous leprosy. Low power magnification.

CLASSIFICATION AND TYPES

Leprosy, supposedly, is caused by the bacillus discovered by Hansen in 1872. This is an acid-fast, curved or straight rod, easily demonstrated in most lepers' eyes at autopsy. Clinically, the disease is usually classified as cutaneous or neural, depending on the structures involved. These types can be studied histologically in the skin and nerves of the adnexa more readily than in the internal tissues of the eyeball. The cutaneous, or lepromatous, type (fig. 1) is characterized by a thinning of the

epidermis, which is pushed outward in places into a nodular protrusion and separated from the underlying leproma by a thin layer of normal appearing connective tissue. The leproma itself is made up of a collection of lymphocytes, fibroblasts and large monocytic cells, with an occasional polymorphonuclear. These large monocytes phagocytose the leprosy bacilli and become swollen, and the cytoplasm appears "frothy," hence the name "foam" cells. These resemble fat cells but usually show a

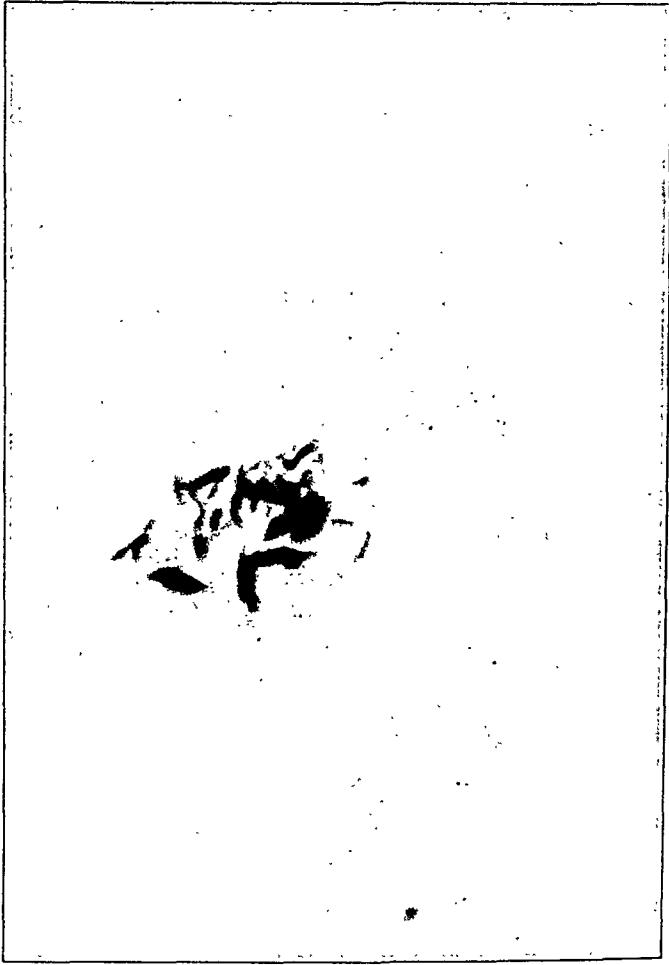


Fig. 2.—Typical leper or "foam" cell. High power magnification, oil immersion lens; $\times 2,040$.

nucleus (fig. 2, photomicrograph of a blood smear contaminated by tissue juice).

The neural lesions (fig. 3) usually do not contain any "foam" cells or bacilli but consist of an infiltration of the perineurium with lymphocytes, a few monocytes and fibroblasts. Eventually fibrous tissue in small clumps is laid down in the perineurium and endoneurium, which destroys the nerve fibers by pressure. There are two additional histo-

logic types in the leprous curriculum, namely, the macular and the tuberculoid, or "reacting macular," type. Because of the fact that true macules are rarely observed on the ocular structures and the latter classification is not universally agreed on by leprologists, these groups will be disregarded. By far the greater percentage of ocular leprosy is of the mixed type. The leprosy in 8.8 per cent of the 350 cases was classified as neural, in 9.7 per cent as cutaneous and in the remainder (81.5 per cent) as of the mixed type.



Fig. 3.—Neural leprosy, showing infiltration and extreme fibrosis of endoneurium. Low power magnification.

OCULAR INVOLVEMENT

Most writers agree on the frequency of ocular involvement in leprosy. Minder¹⁰ found 50 per cent of the eyes he examined affected. Lopez¹¹ stated that all eyes are diseased if one includes the adnexa. DeSilva, in

10. Minder, F.: *Klin. Monatsbl. f. Augenh.* **84**:36 (Jan. 25) 1929.

11. Lopez, E.: *Arch. Ophth.* **18**:404, 1889.

Siam, found ocular complications in 20 per cent of 500 cases. Wood¹² found all persons he examined in South Africa to have ocular complications. Borthen and Lie,¹³ citing Norwegian cases, reported ocular involvement in 75 per cent of cases of the neural type and in 90 per cent of cases of the cutaneous type. Kaurin later reported that between two thirds and three fourths of the patients he examined in Norway had ocular lesions. Chance¹⁴ thought the eye not to be involved until the disease was 2 years old. Neve¹⁵ found 25 per cent involvement in patients from the Kashmir leper asylum. Grossman¹⁶ stated that patients having cutaneous leprosy always have ocular complications, and Rogers and Pinkerton both stated that the eyes are invariably involved if the patient lives long enough. The latter found the eyes of 323 of 363 patients examined to be diseased. This corresponds almost identically with my findings of only 33 of 350 patients with normal eyes, a total of 91 per cent involvement in some form or other.

PORTAL OF ENTRY

Many diverse opinions have existed as to the mode of infection of the eye ever since leprosy was first studied. Some think the bacilli enter the eye externally, from the conjunctiva or episcleral tissues. Others are of the opinion that it is endogenous in origin. The latter view seems to be more plausible and accepted at the present time. It is well known that a bacillemia occurs in many cases of cutaneous leprosy and that bacilli can be found in many of the internal organs at autopsy. These facts are attested to by such men as Honeij,¹⁷ Johansen¹⁸ and Wayson. Axenfeld,¹⁹ in supporting the hematogenous theory, called attention to the arrangement of the vessels at the limbus, stating that most bacilli are found in this region; he cited Lie as declaring that all ocular involvement begins from the angle of the anterior chamber. These views were upheld by Jeanselme and Morax.²⁰ In a recent article Fuchs²¹ stated that he found the primary ocular lesions to be in the anterior part of the ciliary body around the major circle of the iris. The episcleral inflammation,

12. Wood, D. J.: *Brit. J. Ophth.* **9**:1 (Jan.) 1925.

13. Borthen, L., and Lie, H. P.: *Die Lepra des Auges*, Leipzig, Wilhelm Engelmann, 1899.

14. Chance, B.: *Ann. Ophth.* **25**:432, 1916.

15. Neve, A.: *Brit. M. J.* **1**:1153, 1900.

16. Grossman, K.: *Brit. M. J.* **1**:11, 1906.

17. Honeij, J. A.: *J. Infect. Dis.* **17**:376, 1915.

18. Johansen, F. A.: *Am. Rev. Tuberc.* **35**:609 (May) 1937.

19. Axenfeld, T.: *Bacteriology of the Eye*, translated by A. McNab, London, Baillière, Tindall & Cox, 1908.

20. Jeanselme, E., and Morax, V.: *Ann. d'ocul.* **130**:321, 1898.

21. Fuchs, A.: *Klin. Monatsbl. f. Augenh.* **98**:728 (June) 1937.

as in tuberculosis, appeared to him to be more virulent and recent than that in the deeper structures, indicating outward extension. He criticized Uchidas' recent experiments with rats which supported the theory of inward extension on the grounds that the condition is entirely different in human beings. Sticker²² expressed the belief that the primary focus is in the nose and that the eye is secondarily involved from this. Dimitry²³ said the ocular condition does not exist when the nose is free from pathologic involvement. He laid special stress on nasal treatment. On the other hand, because he was unable to find bacilli in end vessels of the iris, ciliary body and retina, Babes questioned a hematogenous origin. Hoffmann²⁴ and Gyotoku²⁵ were agreed on involvement by extension from the conjunctiva and skin of the adnexa.

Much animal experimentation, mostly on rabbits, has been conducted on the transference of human ocular leprosy. Thus Vossium, Neisser, Melcher and Ortman, Ivanow, Campana, Kitasata, Bayon, Zenoni and others claimed to have succeeded in this, while negative results were obtained by Hansen, Vidal, Kobner, Colderaro, Gauchen and several others. Maucione²⁶ in 1924 performed some painstaking experiments along this line, with completely negative results. Santonastaso²⁷ is the latest flatly to deny this possibility.

AGE AND SEX CHARACTERISTICS

The youngest patient examined at the National Leprosarium was 8 and the eldest 77. I found middle-aged persons and those who gave a history of long duration of the disease in general had the most severe ocular complications. Patients who contracted the disease during or beyond middle age seem to be the most immune. However, this was variable because of the still undetermined period of incubation. Seventy-one per cent of the patients examined were male and 29 per cent female. Both sexes were involved alike as to type of the disease.

VISION AND PERCENTAGE OF BLINDNESS

The 350 patients examined represent a total of 698 eyes; 20, or 5.7 per cent of the patients, were blind in both eyes; 29, or 8.2 per cent, were blind in one eye. Eighteen, or 5.1 per cent, had perception of light or perception and projection in both eyes, and 40, or 10.2 per cent, had perception, or perception and projection in one eye. Thus a total of

22. Sticker, G.: *Mitth. u. Verhandl. d. internat. Lepra-Confer. zu Berlin* (pt. 1) 1:99, 1897.

23. Dimitry, T. J.: *Am. J. Trop. Med.* **11**:65 (Jan.) 1931.

24. Hoffmann, W. H.: *J. Trop. Med.* **33**:233 (Aug. 15) 1930.

25. Gyotoku, K.: *Ztschr. f. Augenh.* **63**:254 (Nov.) 1927.

26. Maucione, L.: *Arch. di ottal.* **31**:385, 1924.

27. Santonastaso: *Ann. di ottal. e clin. ocul.* **65**:321 (May) 1937.

29 per cent were blind or had perception, or perception and projection in one eye or in both eyes. Neve,¹⁵ in his series of patients from India, found 13 blind eyes in 80 patients and declared that blindness due to leprosy was more common elsewhere. Van Driel²⁸ estimated only 7 cases of blindness in 1,300 patients examined in Sumatra. Eighty-six, or 24.5 per cent, of the patients examined by me had normal vision in both eyes, and 58, or 16.5 per cent, had normal vision in one eye, making a total of 41 per cent with normal vision in one or both eyes.

RACIAL IMMUNITY

Fifty and two-tenths per cent of the patients were white Americans, 25.1 per cent were Mexicans, 9.1 per cent were Negroes and 3.4 per cent each were Italians, Chinese and Filipinos. The remainder constituted small numbers of Jewish, Cuban, Spanish, Puerto Rican, Hawaiian, Portuguese, Japanese, Indian, Hungarian, Hindu and Finnish patients

TABLE 1.—*Data on Vision and Percentage of Blindness*

Total number of eyes examined	698
Total number of patients blind in both eyes	20, or 5.7%
Total number of patients blind in one eye	29, or 8.2%
Total number with perception of light or perception and projection in both eyes	18, or 5.1%
Total number with perception of light or perception and projection in one eye...	40, or 10.2%
Total number blind or having perception or perception and projection in one eye or in both eyes.....	29 %
Number with normal vision in both eyes.....	86, or 24.5%
Number with normal vision in one eye.....	58, or 16.5%
Total number with normal vision in one eye or in both eyes.....	41.0%

in order. It has been noticed by officers of the leprosarium that the Mexican and Filipino population seem to be less susceptible to ocular disorders. My check on this revealed that 22.7 per cent of inmates having normal vision were white, while 36.7 per cent were Mexicans. Of the total number having apparently normal eyes, 6.2 per cent were white and 11.7 per cent were Mexicans. It is difficult to ascribe a cause for this so-called immunity. Certainly it is not a pigment factor, as the Negro seems to be extremely prone to ocular complications. Most of the Mexican patients observed here were fairly young; possibly the disease is discovered earlier in persons of this nationality, and they are, unlike the white man, satisfied to be segregated before leprosy has invaded the eye.

INTRAOCULAR TENSION

Tension was recorded with the McLean tonometer for 318 patients. Only 1 patient showed an elevation of tension, and this responded fairly well to miotics and exhibited no form field or scotoma defects. Sixteen patients had secondary glaucoma, 2 with involvement of both eyes. One

28. Van Driel: *Geneesk. tijdschr. v. Nederl.-Indië* 62:770, 1922.

wonders why there is not more secondary glaucoma after observing the widespread involvement of the iris in leprosy. The reason, of course, as Kirwan²⁹ has stated, must be because of the extreme atrophy and fibrosis of most of these irides and their pectinate ligaments. Wood declared that because of this condition it is almost impossible to do a satisfactory iridectomy in the majority of the cases of well marked involvement. DeSilva also found few instances of primary glaucomas in a series of patients examined in Ceylon.

ADNEXA AND EXTRAOCULAR MUSCLES

Multiple and diverse lesions and deformities are found on the adnexa in leprosy. Most prominent and early among these is the nodular hypertrophy of the brows and lids in the cutaneous types, occurring, of course, in conjunction with the lesions on the face. In certain cases one also sees simple hypertrophy without nodule formation. Madarosis, or absence of hair, is present in nearly all these cases and may be partial or

TABLE 2.—*Data on Racial Immunity*

White Americans	50.2%
Mexicans	25.1%
Negroes	9.1%
Italians, Chinese, Filipinos.....	3.4% each
White Americans with normal vision.....	22.7%
Mexicans with normal vision.....	36.7%
White Americans with apparently normal eyes.....	6.2%
Mexicans with apparently normal eyes.....	11.7%

complete. When it is partial on the brows, the hairs are lacking externally (fig. 4). Approximately 47 per cent of the patients had hypertrophy or nodule formation on the lids or brows or both, with partial or complete madarosis. Twenty-three and seven-tenths per cent had madarosis without hypertrophy or nodule formation. The latter group for the most part were persons with neural and old "burned out" cutaneous leprosy.

It was my impression that most of the deep or protected tissues around the eye are fairly immune to leprous invasion. The officers of the leprosarium have noted that lesions on the hand do not progress as rapidly when the patients protect them with gloves. So it is with the eye. In spite of the fact that 17 cases of strabismus were observed, I think the incidence of invasion of the well shielded extraocular muscles with their nerve supply is low. In 12 of the 17 cases the strabismus was of the divergent variety, and for the most part in blind, phthisic eyes. The facial nerve, in fact, seemed to be the only cranial nerve markedly affected. This could be explained by its wide superficial

29. Kirwan, E. O.: *Far East. A. Trop. Med., Tr. Seventh Cong. (1927) 1*: 289, 1928.

distribution. This protection immunity, of course, is not true of the uveal tissues. One is forced to explain the frequency of anterior uveitis by the abundant vascular supply.

Another interesting illustration of the influence of protection is that of a Negro (fig. 5) whose palpebral openings were narrowed considerably by contraction from keloids. In spite of a good amount of mixed generalized cutaneous leprosy, the corneas of this man were only slightly involved. Valle³⁰ recently emphasized the importance of protecting the eye as a prophylactic measure in leprosy. Owing to involvement of the

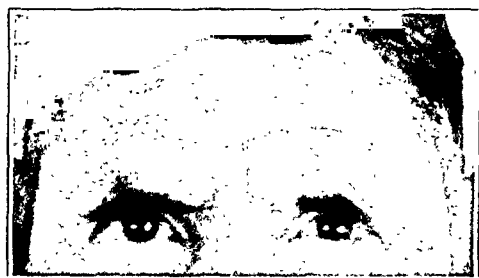


Fig. 4.—Partial madarosis.

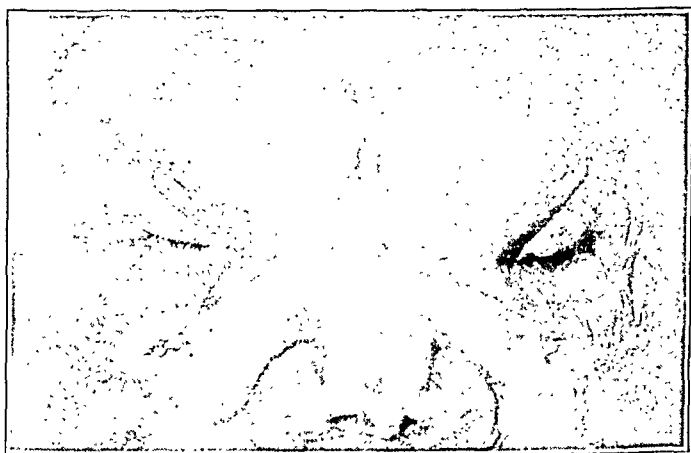


Fig. 5.—Protective influence of narrowed palpebral openings on the cornea.

facial nerve, 15.7 per cent of the patients had lagophthalmos of one lid or of both lids. There were only 9 cases of marked ectropion but many more of the milder type of this condition. King,³¹ in describing cases he observed in England, stated that paralysis of the facial nerve is always preceded by blinking and fibrillation.

Coexistent with, or shortly after, the development of changes in the nasal bone, one sees obstruction of the lacrimal canal, which often results

30. Valle, S.: *Rev. de oftal. de São Paulo* **5**:3 (Sept.) 1936.

31. King, E. F.: *Brit. J. Ophth.* **20**:561 (Oct.) 1936.

in suppurative conditions of the tear sac. The infection is probably an ascending one directly due to bony necrosis. There were 8 cases of dacryocystitis and 3 cases of chronic external fistulization of the lacrimal sac.

An interesting feature of the adnexa is the prevalence of xanthelasma among lepers. In a recent survey, Dr. Ralph Hopkins, the dermatologist at the leprosarium, found 10 per cent of patients to have this condition. In a few of the patients I examined xanthelasma was found to be superimposed on the leprous nodules on the lids and was occasionally found all over the body. The blood study of the patients with this condition revealed a disturbed cholesterol metabolism. This relationship was presented by Hopkins, Black and Ross.³²

Another important observation on the lids consists of a so-called "immune area" in the region of, and internal to, the folds in the upper and

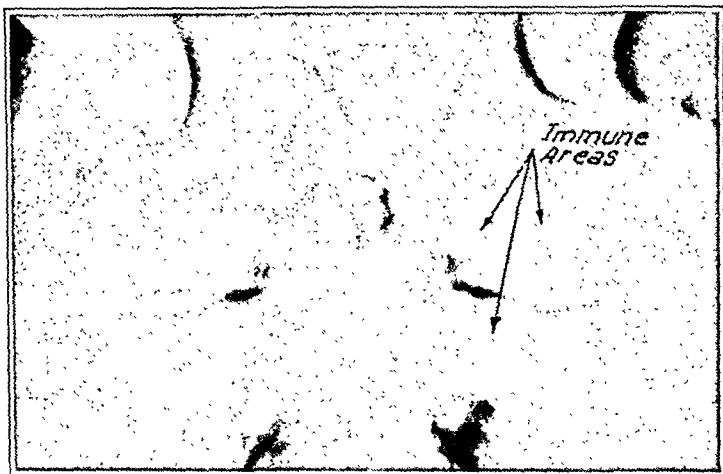


Fig. 6.—Immune areas on the adnexa.

lower lids (fig. 6), first reported several years ago by Hopkins, Denney and Johansen.³³ This area, of course, is well protected by bony structures and infolding of the skin and again reemphasizes the importance of protection.

CONJUNCTIVA

One hundred and eighty persons, or a little more than half the patients examined, had conjunctivitis. The infection in only a few cases, however, failed to respond to the usual treatment and could be classified as being of leprous origin. Pinkerton likewise expressed the belief that

32. Hopkins, R.; Black, S. H., and Ross, H.: Xanthelasma and Leprosy, *Arch. Dermat. & Syph.* **39**:239 (Feb.) 1939.

33. Hopkins, R.; Denney, O. E., and Johansen, F. A.: Immunity of Certain Anatomic Regions from Lesions of Skin Leprosy, *Arch. Dermat. & Syph.* **20**: 767 (Dec.) 1929.

the incidence of leprous conjunctival involvement is nil except at the limbus. In a series of 149 patients he found 45 to have injected conjunctival membranes, none of which he thought to be leprous. The conjunctiva is subject to invasion in a threefold manner: externally from the skin and nasal secretions; internally from the episcleral tissues, especially at the limbus, and again from the blood stream. In spite of this, it remains one of the fairly immune ocular structures. The bulbar conjunctiva does, of course, participate in the usually severe leprous infiltration at the limbus, but I believe this may be due to its close approximation to the episcleral tissues and Tenon's capsule in this location. This sparing of the conjunctiva seems rather unusual, since Fuchs found that in 66 per cent of cases the tears bathing the conjunctiva contain bacilli. Here again protection may play a role, as the greater part of the conjunctiva is well covered. A great many of the conjunctivitis observed were undoubtedly caused by the too promiscuous use of atropine. The conjunctiva of the leprous eye seems to tolerate a 0.33 per cent solution of scopolamine hydrobromide much better than atropine.

OPHTHALMOSCOPIC EXAMINATION

Because of corneal and lenticular opacities as well as pupillary exudates, the fundi of only 243 patients could be observed, and the surprising thing encountered was that in 201 of these the fundus in each eye was absolutely devoid of lesions, disregarding, of course, vascular changes of the arteriosclerotic variety observed in many of the older persons. Even in many cases of advanced corneal and iritic involvement the retina appeared normal. There were 14 cases of choroiditis or chorioretinitis, 13 of well marked opacities of the vitreous, 6 of optic neuritis, 5 of atrophy of the optic nerve and several in which there were medullated nerve fibers, retinal hemorrhages and a mild degree of cupping of the optic nerve. In not 1 of 28 sectioned eyes was I able to find leprous changes or bacilli in the retina or optic nerve. Lie found bacilli in only one section of an optic nerve in a series of eyes he examined, and this was not accompanied by any inflammatory reaction.

This frequency or infrequency of changes in ocular neural tissues has been discussed pro and con ever since Trantus reported having found retinal lesions ophthalmoscopically in 68 per cent of a series of patients he examined. Later, he ³⁴ cited Bistis ³⁵ as having confirmed his work and stated that Rubert ³⁶ had found retinal changes in 23 per cent of 202 lepers. Trantus reported that the lesions increased and are more frequent in the region of the ora serrata and laid special stress on digital

34. Trantus: *Arch. d'opht.* **32**:193, 1912.

35. Bistis, J.: *Centralbl. f. prakt. Augenh.* **23**:328, 1899.

36. Rubert, I. U.: *Diseases of the Eyes in Leprosy*, Yuryev, Schnakenbury, 1903.

pressure to bring them in view. It was my experience that not over 15 per cent of these eyes would dilate sufficiently to enable one to view the most anterior parts of the retina, even with digital pressure or any other type of manipulation. It is true that most retinal changes if present should be confined to the anterior portion as this is the part of the eye most affected, but Trantus' findings have been criticized by many subsequent observers. Parsons in his text on the pathology of the eye stated that as a rule leprous retinal lesions are slight. I believe that the small percentage of anterior retinal lesions present are probably primarily choroidal.

The reported similarity of these leprous retinal lesions to those of syphilis and tuberculosis could not be verified by my observations. The small number of lesions observed by me resembled no particular retinal entity. Syphilis is extremely hard to prove in lepers, since over 50 per cent of leprous serums give positive Wassermann, Kline and Kolmer reactions. However, I did place a small number of patients on arsenical

TABLE 3.—*Results of Ophthalmoscopic Examination*

Number of fundi observed	243
Number of normal fundi	82.7%
Number of cases of choroiditis or chorioretinitis.....	14
Number of cases of optic neuritis	6
Number of cases of atrophy of the optic nerve.....	5

preparations as a therapeutic test, without results. Van Poole,⁹ in his excellent thesis on leprosy and tuberculosis of the eye, concluded that these diseases rarely cause lesions of the posterior segment. He attributed an allergic causation to the optic neuritis he found in 49 of 206 cases of leprosy. This seems to me to be fairly logical, but the optic neuritis in the few cases observed by me was not of the transitory type which he described.

CORNEA

The cornea is the most vulnerable ocular tissue to leprosy and enables one to see and study an involvement analogous to that of the skin and nerves. Elliot³⁷ summarized the fourfold damage to the cornea in the following manner: (1) loss of sensitiveness, (2) loss of nutritional control, (3) exposure due to paralyses of the muscles and (4) diminution of the lavage control.

The primary and most common change noted by me was the infiltration in the deep layers of the stroma (fig. 7). This almost always starts from above, but occasionally it starts from the temporal side; as seen with the slit lamp, it resembles a syphilitic interstitial infiltration, having a dull gray color and a rather amorphous structure with poor outline. When fairly advanced, a vascular net develops and follows the infiltration.

37. Elliot, R. H.: *Tropical Ophthalmology*, London, H. Frowde, 1920.

The second most common lesion was the 'superficial leprotic keratitis which many observers have described as the primary change but which in many cases I observed followed the interstitial infiltration. This usually also starts from above and blankets the deep haze with a network of pinpoint white dots, often referred to by writers as "chalk dust" or "flour dust." All these minute superficial areas are surrounded by a grayish infiltration, and the corneal tissue between them is dull and lusterless. The entire area is sharply demarcated and when well advanced becomes vascularized and takes on the appearance of pannus formation. Microscopically (fig. 8) these superficial lesions resemble cutaneous leprosy. The epithelium is usually intact but irregular. Bow-

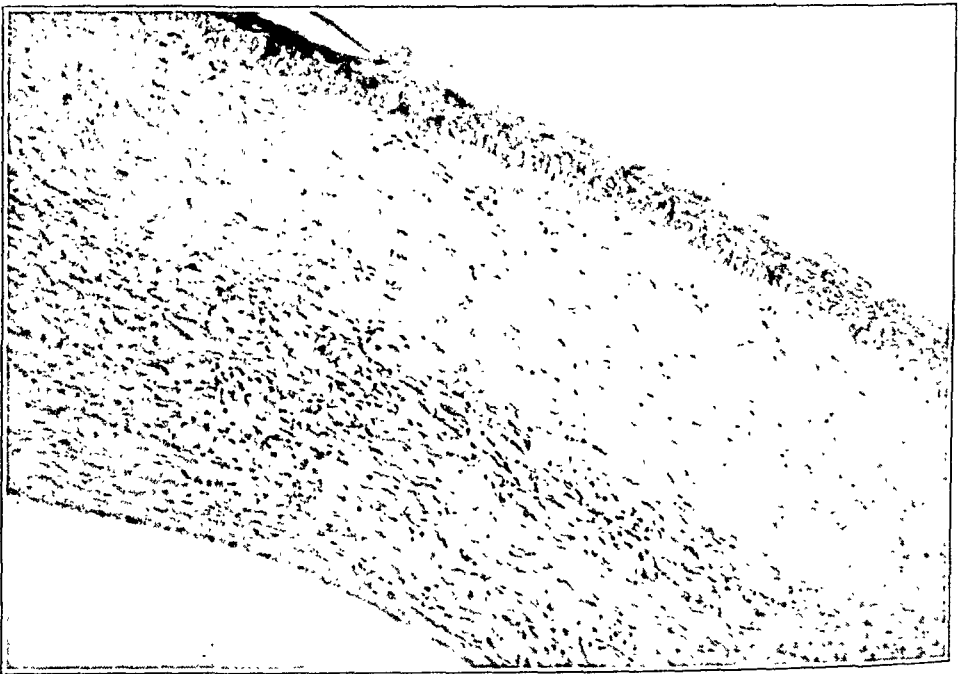


Fig. 7.—Deep corneal infiltration. Low power magnification; $\times 140$.

man's membrane is either absent or pushed up against the epithelium. It corresponds to the thin line of connective subepithelial tissue in the cutaneous lesions. The lamellae are separated, and there is hyperplasia of fixed and wandering corneal cells, which collect in groups under Bowman's membrane to form a typical leproma. Bacilli, both extracellular and intracellular, are numerous as observed with the acid-fast stain. Descemet's membrane seems to be unaltered even in cases of advanced leprosy. Sogunama³⁸ stated that the first changes take place under Bowman's membrane at the perforations of the nerves and that the interruption of this structure limits the lesion. • There was a deep or

38. Sogunama, S., and Sogunama, H. M.: *Arch. f. Augenh.* **77**:227, 1917.

superficial involvement of the cornea in 289 cases. Bacilli were found in 18 and leprous lesions in 22 of 28 sectioned corneas.

In a number of corneas with these deep and superficial changes, beaded corneal nerves were observed with the high power objective of the slit lamp at the limbus. These tiny nodes or enlargements on the shaft of the nerve were described by Minder to be a round cell infiltration of the endoneurium and sheath of Schwann. They seemed to me to be

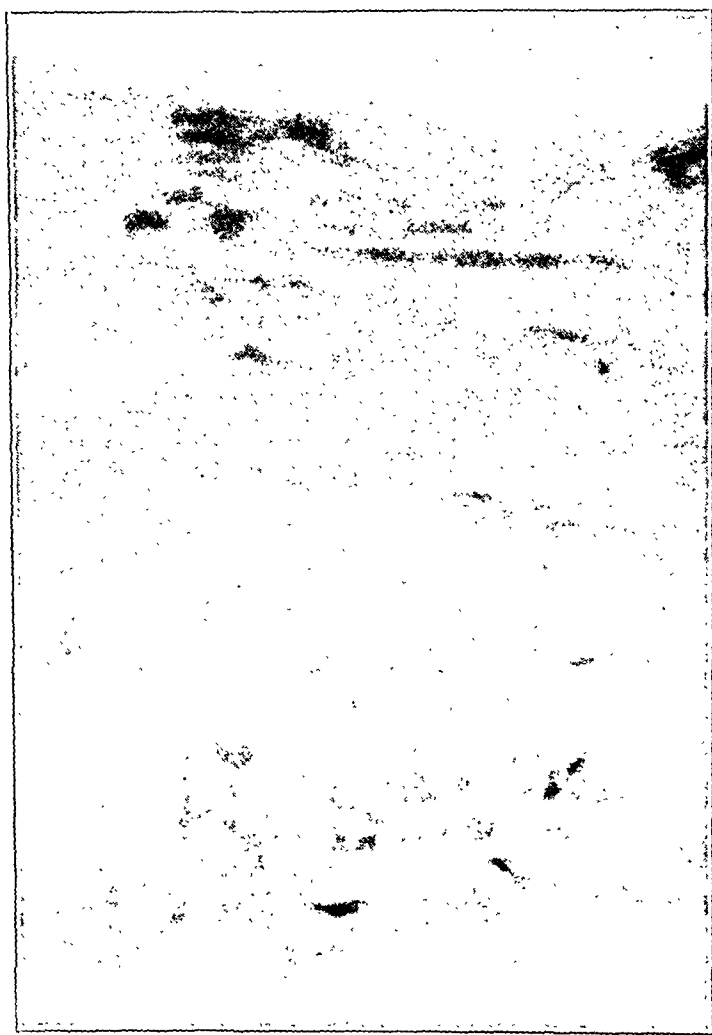


Fig. 8.—Infiltration and bacilli in the superficial corneal layers. High magnification, dry lens; $\times 760$.

analogous to generalized neural involvement. Only in cases of well advanced superficial keratitis did I find corneal anesthesia to be present, and superficial ulceration was not nearly as prevalent as one would expect. The most intense microscopic corneal leprous lesions are found at the limbus. Here diffuse cell infiltration produces the macroscopic scleral roll, which may circumscribe the cornea (fig. 9) or may be well localized.

Gabriélidès,³⁹ Sauvinau and Morax and, more recently, Pillat⁴⁰ and others have laid special stress on the diagnosis of ocular leprosy by examination of superficial corneal scrapings. Undoubtedly bacilli are present in this location if at all. However, I believe that a competent dermatologist can usually make a diagnosis of leprosy several years before a diagnosis is made by this means.

The most marked and disfiguring type of gross corneal lesion was the immense leproma found in 3 cases. This growth had a smooth, glistening appearance and was found to cover the entire cornea (fig. 10). At first I thought it was extracorneal, and even when it was removed it dissected easily; in fact, it almost peeled from the corneal surface. On microscopic section, however (fig. 11), this gave the appearance of a massive intracorneal leproma. The epithelium was greatly hypertrophied and almost cornified. Bowman's membrane was still present; it was pushed up against the epithelium and separated the

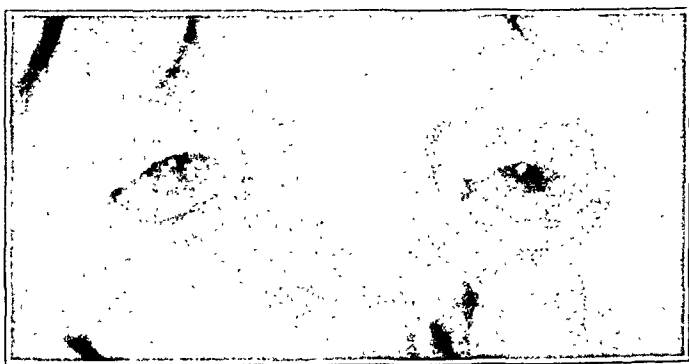


Fig. 9.—Corneoscleral roll.

latter structure from an intense leprous infiltration, in which a good number of bacilli and cells were found as well as a fair amount of fibrous tissue.

Ophthalmologists studying leprosy have often been baffled at finding lepra bacilli in apparently normal corneas and still have reached no definite conclusion as to a solution to this phenomenon. Some have used the term "host resistance"; others, "attenuated bacilli," and Fuchs recently used the phrase "tissue behavior" to explain this strange condition. I think one must again turn to the dermatologists for the most plausible solution. They state that they find bacilli in normal appearing skin and that these organisms, under an allergic influence and without any increase in their number, may set up a reaction which will vary in

39. Gabriélidès, A.: Arch. d'opht. **34**:439, 1914.

40. Pillat, A.: Leprosy Bacilli in Scraping from Diseased Cornea in a Leper and Comments on Keratitis Punctata Superficialis Leprosa: Report of a Case, Arch. Ophth. **3**:306 (March) 1930.

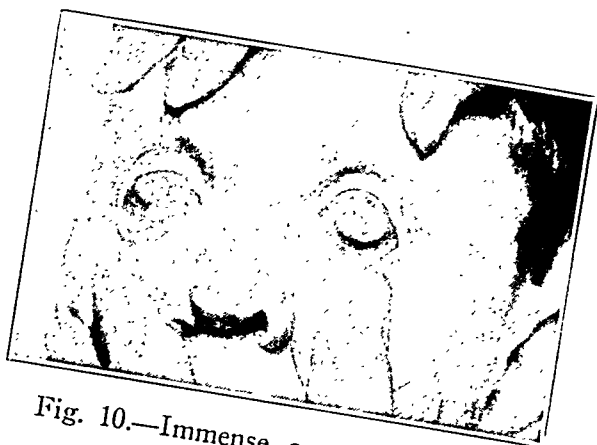


Fig. 10.—Immense corneal leproma.

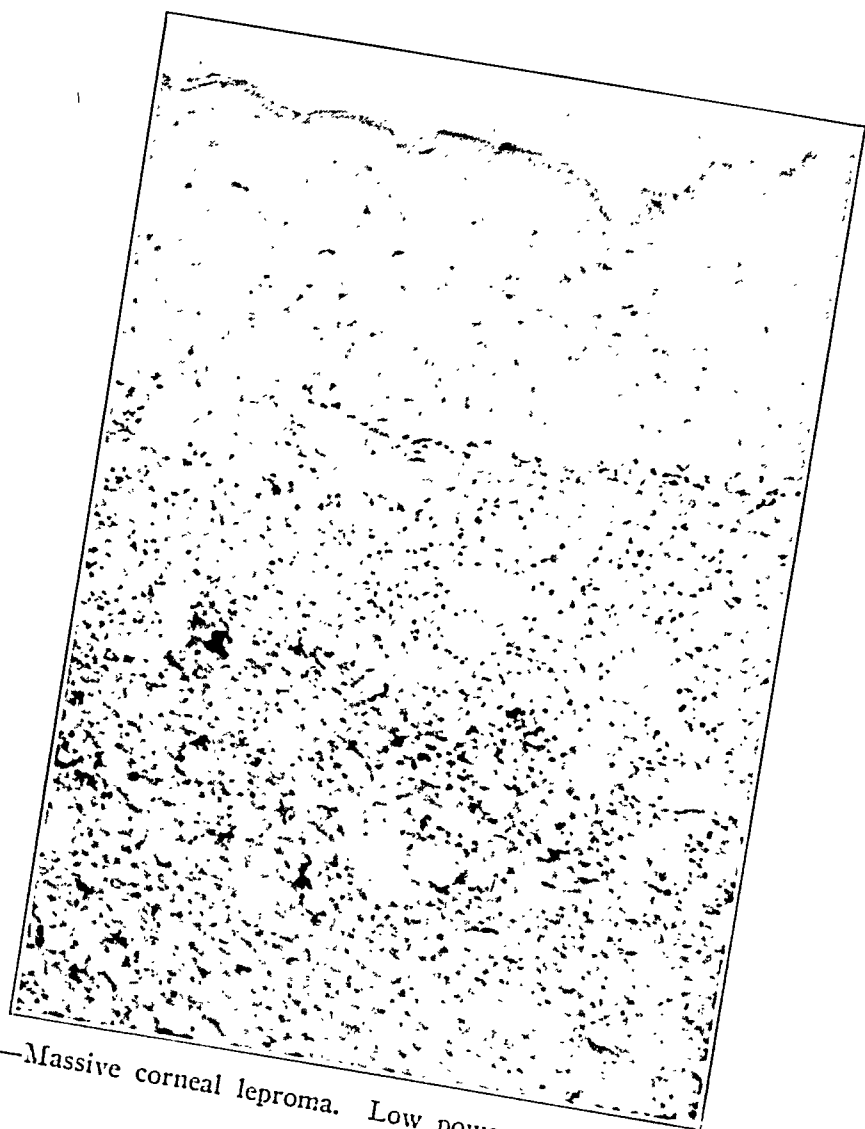


Fig. 11.—Massive corneal leproma. Low power magnification; $\times 140$.

intensity and after which they will be destroyed. These temporary corneal reactions in leprosy may be severe or mild, and I can think of no better way to account for them. Unfortunately, I had the opportunity of subjecting to slit lamp examination only a few corneas which were later sectioned, and these were all involved in some manner.

Another question frequently raised concerns the manner in which these nonmotile bacilli find their way into an avascular structure such as the normal cornea. Lie stated the bacilli come in by the lymph and are then taken up by the corneal cells and that all involvement starts from the angle of the anterior chamber. Axenfeld declared they are carried to the limbus by the cells and "diffuse themselves to the interstitium," where they may remain indefinitely. My impression is that leprosy bacilli are transported by the blood stream to the ciliary body by the major circle of the iris and then are disseminated throughout the cornea both by wandering cells and by the lymph stream. Possibly they may reach the aqueous through the epithelium of the ciliary body and thence be carried to the cornea through Fontana's spaces.

SCLERA

Leprous lesions of the sclera, as in tuberculous lesions, are almost always confined to the corneoscleral junction. Here one finds active and intense lesions involving for the most part the episcleral tissue. Leprosy cells and bacilli are usually plentiful in this region, and the inflammatory reaction may be localized or pericorneal. Deep scleral lesions, however, are rare. In not 1 of 28 sectioned eyes was I able to find lesions or bacilli in the deep, densely packed connective tissue of the sclera proper.

IRIS

Subject to intermittent attacks, the iris shows all gradations of involvement from a mild degree of swelling with obliteration of the anterior pattern and ectropion of the pigment frill to almost complete fibrosis and atrophy. Posterior synechia, anterior capsular pigment deposits and polycoria were frequent. Cysts of the posterior epithelium were observed in 3 cases. Iridectomy performed in cases of advanced leprosy usually encountered a friable iris, which left its pigment epithelium adherent to the lens capsule. Bacilli and lesions were found in 13 irides of the sectioned eyes. These were usually located near the base of the iris, close to the posterior pigment epithelium.

The most classic and striking lesion in leprosy of the iris is the leproma, or nodule, seen in 52 cases. With the corneal microscope they appeared as white globular bodies usually enmeshed in the iris tissue, but at times they were observed to protrude on the surface and almost appeared pedunculated. The three different positions that they occupy

are shown in figure 12. Most often they occur around the pupil from the rim back to the collaret (fig. 12 *A*). Again they are seen diffusely throughout the anterior mesodermal layer (fig. 12 *B*). Occasionally they are observed at the peripheral border, adjacent to a corneoscleral lesion (fig. 13 *C*).

Meller ⁴¹ expressed the belief that lepromas of the iris are rare and cited Borthen as having found only 4 or 5 in a series of 250 cases. Pinkerton expressed doubts as to whether these are true lepromas, but Wood declared them to be distinctly pathognomonic of leprosy, and Peter ⁴² concluded that they were nodules from their close microscopic resemblance to cutaneous lepromas. I have never seen any postinflam-

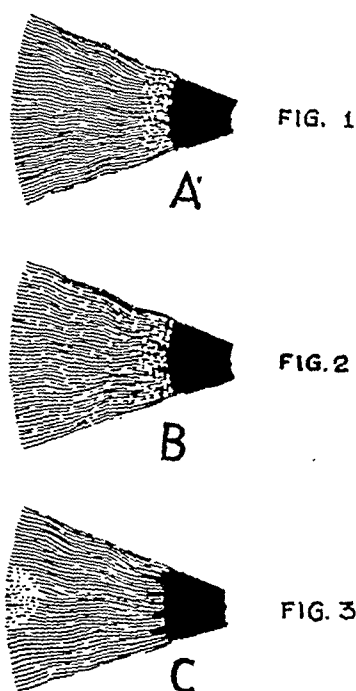


Fig. 12.—Position of iris nodules.

matory changes resembling these lesions and could call them nothing but lepromas or nodules.

CHOROID AND CILIARY BODY

Although atrophy and hyalinization of the ciliary body, with its processes, is frequent in sections of leprosy eyes, one does not as a rule see signs of acute inflammation and dense cellular infiltration in this structure. There is usually a scattering of chronic inflammatory cells and bacilli around the major arterial circle and stromal parts, and "foam" or leper cells are fairly infrequent (fig. 13).

41. Meller, J.: *Klin. Monatsbl. f. Augenh.* **43**:66, 1905.

42. Peter, L. C.: *Arch. Ophth.* **53**:258 (May) 1924.

The ciliary muscles are thin, and the radial portion of Brucke's muscle seems in many instances to limit the posterior extension of cells and bacilli. Single bacilli can be traced, however, along the pigmented posterior fibers of this muscle into the choroid. By far the greatest amount of choroidal involvement was observed in the anterior segment, and, like the involvement of the ciliary body, it was of a chronic nature. This seemed to bear out Fuchs's contention, viz., that the bacilli seem to be more virulent and destructive when observed in the more external ocular tissues, such as the cornea or episclera, thus appearing to favor outward extension. Leprous lesions were found in the ciliary body of 19 of the 28 eyes examined and bacilli were found in 14. Lesions occurred in the choroid of 15 eyes and bacilli in the choroid of 9 eyes.

LENS AND VITREOUS

• No infiltration of bacilli or cells was found in either the lens or the vitreous. Opacities of the lens occurred in 103 cases, but for the most



Fig. 13.—Infiltration in the angle of anterior chamber. Low power magnification; $\times 140$.

part they resulted from the numerous deposits on the anterior capsule. Few opacities of the senile variety were observed.

METHODS AND RESULTS OF TREATMENT

Various methods were used in carrying out treatment of the ocular conditions. Some of the drugs had been used often before in treating general leprosy, but the effect of only a few has been noted on the eye. In instituting treatment of any type of leprosy there are two factors in the patient's general attitude which impresses one. The first is his reluctance to accept anything new or unusual. The reason for this, of course, is that so many remedies have been tried over a period of years that the patients merely believe the treatment to be another experiment. They also have a feeling that the treatment, if new and

untested, may make their condition worse. Secondly, most patients are loath to continue any certain treatment over a long period. Immediate result is the desirable thing.

The patients who volunteered for treatment were divided into three main groups: (1) those with old corneal infiltrates and opacities, (2) those with more or less of a chronic uveitis or iridocyclitis (most of these, of course; also had corneal involvement) and (3) those with an acute or rapidly advancing process.

For the first group four ingredients were used locally, namely, ethylmorphine hydrochloride, chaulmoogra oil, thyroxin and an ointment containing quinine bisulfate. Each form of treatment was started with 20 patients, but many discontinued their visits during the three month period of treatment. The results of this procedure were as follows:

Ethylmorphine hydrochloride was used three times a week in a 10 per cent solution, after the concentration of the solution had been increased gradually up to this dilution. Eleven patients completed the treatment. Three of these said their vision was better. However, 6

TABLE 4.—*Involvement Found in Twenty-Eight Eyes Sectioned*

Location	Lesions Found	Bacilli Found
Cornea.....	22	18
Sclera.....	None	None
Iris.....	13	13
Ciliary body.....	19	14
Choroid.....	15	9
Retina and optic nerve.....	None	None

actually showed a slight increase in visual acuity, 2 in both eyes and 4 in one eye. Slit lamp examination after treatment, however, showed little change in any of the corneal opacities.

Chaulmoogra oil was used in a 50 per cent solution with castor oil. This was extremely irritating to some patients and had to be preceded by the use of a 0.5 per cent solution of pontocaine hydrochloride. Leprologists in the United States are not nearly as enthusiastic about chaulmoogra oil in the treatment of generalized leprosy as the Europeans. Nevertheless, about 90 per cent of the patients here take the drug by mouth. Approximately 28 per cent received it by vein regularly and about 10 per cent irregularly. In this group vision was improved slightly in 2 of 10 patients who completed the treatment, but again slit lamp examination revealed little change.

Thyroxin (Squibb) was used according to Jameson's formula; that is, 1 drop of a solution containing 2 mg. per cubic centimeter was instilled twice daily in each eye. Seventeen patients received the drug, and 11 of them reported better vision, but only 4 had better sight as indicated with the test card after the treatment. Slit lamp evidence

was more convincing in this group, however, especially concerning the interstitial infiltration, which in many cases seemed to be slightly clearer after the treatment.

An ointment containing quinine bisulfate in a 2 per cent concentration was used as advised by Selinger for 18 patients. The ointment was instilled twice daily. Of 9 who were of the opinion their vision was bettered, there were 4 who were actually improved according to the test card. But here again the corneal examination seemed to favor a larger percentage of improvement than that which was obtained.

To 13 patients with chronic ocular leprosy, for the most part consisting of a long-standing uveitis and corneal scarifications, calcium gluconate was given orally as advised by Theobald, 1 drachm (3.7 cc.) three times a day. Some of the patients in this group were unable to understand how oral medication could be beneficial to the eye. Consequently drops of glycerite of boroglycerin were used daily, mostly for a psychic effect. Although little objective improvement could be noted in this group, 5 patients thought their sight was helped, and in 8 the vision was actually improved slightly, in 2 in both eyes.

Two forms of foreign protein therapy were used in the treatment of 22 patients with acute or rapidly advancing ocular leprosy; at the same time they were being treated symptomatically with the usual heat, salicylates and cycloplegia. The first preparation, erysipelas and prodigious toxins (Coley's) (Parke, Davis & Co.), was discontinued after it was used on several patients, as it produced terrific localized cutaneous reactions. Triple typhoid vaccine (Mulford), however, in general produced gratifying results in 19 cases. This, of course, was administered intravenously, and I found it was necessary in most cases to give 50,000,000 killed organisms in order to obtain a good reaction. While this, especially in the cases of cutaneous leprosy, did cause some generalized cutaneous flare-up, the patients felt improved in every way at the termination of the shock. Eleven of the 19 patients reported relief from their ocular distress following injection of triple typhoid vaccine, and this was in direct confirmation with the objective findings.

Because of the similarity of ocular leprosy to certain phases of syphilis and also because of the results Lucic and others obtained in treating patients with nonspecific uveitis with arsenical preparations, 8 patients who did not respond well to protein shock were given intravenous injections of neoarsphenamine in 0.3 to 0.45 and 0.6 Gm. doses. These patients all had positive Wassermann, Kline and Kolmer reactions, which, as stated before, means nothing in leprosy. The results of this treatment were rather disappointing, 3 patients having such severe cutaneous and systemic reactions that further treatment was discontinued. The other 5 did not seem to be aided in any way.

Schnaudigl, Hoffmann²⁴ and Kupffer⁴³ have reported amazing results with gold salts in ocular leprosy. Ten of the patients with the acute type of lesions, who failed to respond to foreign protein or to arsenical preparations, were started on a series of intravenous injections of gold sodium thiosulfate-Abbott, being given four injections of the 10 mg. dose and the dose then being increased to 25 mg. twice weekly. Although this routine was carried out for only about two months, which according to Benedict is not of sufficient duration, no definite improvement could be observed during this time and only 1 patient thought he had been helped. Two severe systemic reactions were obtained.

Because of the similarity of many phases of ocular tuberculosis and ocular leprosy, I thought that it would be interesting to try tuberculin in a group of these patients. Although it is thought that leprosy

TABLE 5.—*Results of Treatment*

Agent	No. of Patients Completing Treatment	No. of Patients Reporting Improvement	No. of Patients with Visual Acuity Actually Improved	Results of Objective Examination After Treatment
Ethylmorphine hydrochloride, 10%.....	11	3	6	—
Chaulmoogra oil.....	10	1	2	—
Thyroxin.....	17	11	4	+
Quinine bisulfate ointment.....	18	9	4	+
Calcium gluconate.....	13	5	8	—
Erysipelas and prodigiosus toxin (Coley)....				
Triple typhoid vaccine.....	19	11	..	+
Nearsphenamine.....	8	0	0	—
Gold sodium thiosulfate.....	10	1	0	—
Tuberculin.....	10	0	1	—

skins are desensitized and react atypically to tuberculin, 11 patients with a variety of ocular lesions were tested intradermally with purified protein derivative (Parke, Davis & Co.). One severe positive reaction with the second dilution (0.005 mg.) was obtained in a patient who had little leprosy and who later was found to have lesions in the upper lobe of the lung on roentgenographic examination. The remaining 10, who gave no diagnostic reaction to either dilution, were started on a treatment of tuberculin Denys, beginning with 0.00001 mg. Absolutely no change could be detected by me in the ocular lesions of these patients as treated with this form of tuberculin over a period of three months. However, 1 patient did have a slight improvement of vision in one eye with the test chart.

SURGICAL PROCEDURES

In considering surgical treatment of the leprosy eye, there are two foremost questions which enter the surgeon's mind and which concern

43. Kupffer, A.: *Med. Klin.* **23**:364 (March 11) 1927.

about 80 per cent of the patients. First, is it possible to remove a part of the iris behind any portion of the cornea which happens to be clear and, if this is possible, will this artificial pupil remain clear and how long? Second, is it possible to improve the patient's cosmetic appearance by means of any surgical procedure on the lid? The first consideration is paramount, as there is nothing known which will hinder the further opacification of the cornea. Of course, if the cornea is greatly involved, the iris has probably also been mutilated by the progress of the disease, and iridectomy attempted on this type of eye is not successful. The tissue is so fibrotic and friable that it pulls away in pieces and its posterior epithelium clings to the capsule of the lens or the iridectomy opening will quickly fill in again with exudate or the clear cornea in front will soon become clouded. Hence despite the fact that iridectomy is probably the most common operation performed on leprosy eyes, it still remains one of the most unsatisfactory. Of the 30 operations which I performed, there were 8 iridectomies, and only 3 resulted in better subsequent vision. There were 9 plastic operations performed on lids, most of which gave satisfactory results. The remaining operations consisted of several cataract extractions, operations for pterygium, enucleations and synechotomies and one operation for squint and one discission. At one plastic operation a mucous membrane graft from the lip was successfully implanted in the lower cul-de-sac to correct an almost complete ankyloblepharon. No postoperative intraocular or extraocular infections occurred. It has been noted by many that in all phases of surgical intervention on leprosy postoperative infection is rare. The Hansen bacillus seemingly bestows immunity on the patient.

SUMMARY AND CONCLUSIONS

Findings in 350 cases of ocular leprosy are tabulated. Blindness due to leprosy is common in the United States. Twenty-nine per cent of the patients were blind or nearly blind in one eye or in both eyes, and 91 per cent had ocular involvement in some form or other by the disease.

Mexicans are not so susceptible to ocular leprosy.

Glaucoma is uncommon.

Protection of the leprosy eye in some form or other is an important prophylactic measure.

There are "immune" protected areas on the adnexa. Xanthelasma is common on the adnexa.

Lesions in the retina, optic nerve and lens are rare. The cornea is the most vulnerable ocular tissue and is probably invaded through the blood stream primarily.

Quinine bisulfate ointment and thyroxin administered locally give fair results in clearing the corneal infiltrates. Ethylmorphine hydrochloride

ride and chaulmoogra oil are of no avail. Protein shock gives good results in the treatment of the acute lesions.

The leprous eye, while not being responsive to intraocular surgical treatment, still is not prone to postoperative infection.

424 Hamm Building.

The medical director, H. E. Hasseltine, the commanding officer and other resident officers of the Public Health Service at the National Leprosarium, Carville, La., cooperated in the preparation of these patients.

ABSTRACT OF DISCUSSION

DR. GIDEON M. VAN POOLE, Honolulu, Hawaii: As one who has been interested in the subject of leprosy for more than a decade, I can testify not only to the completeness of the paper but to the soundness of its conclusions. The subject of leprosy holds a fascination for the physician which is difficult to resist. The lack of knowledge concerning the exact causal relation and transmission of leprosy and the inability to supplement facts with experimental observation as well as other difficulties lend the disease its fascination and make any additional contribution a difficult but worthwhile effort.

Dr. Prendergast's material is extensive, and his series of 28 eyes studied pathologically is large indeed and only rarely equaled in the literature. The question of the portal of entry has been dealt with completely, but it may be of interest to add the ingenious theory of S. Valle, of Brazil, who believes that most of the peculiarities of leprous ocular involvement are accounted for by the rich anastomoses between the anterior ciliary arteries and the posterior conjunctival vessels, which in turn bring in bacilli from the lids, superciliary region and forehead.

The important observation on the inverse relation between ocular protection and ocular leprosy is well worth noting and remembering by all who deal with the disease.

I am gratified to learn that Dr. Prendergast endorses my findings and views on the question of leprosy of the posterior segment, but I wish to emphasize that the transitory optic neuritis, or pseudoneuritis, I have observed occurred in conjunction with attacks of acute leprous reaction. When I say pseudoneuritis I get into trouble, because pseudoneuritis is known to be a congenital condition found in hyperoptic persons with a narrowing of the scleral canal, and in that condition bilateral elevation of the disk of about 1 or 2 diopters occurs. There is no interference with vision, and that is exactly what I found in these cases. In true neuritis, which is usually unilateral and comes on suddenly, there is a loss of vision, and both hemorrhages and exudates are found in the fundus. Hemorrhages are not found in the fundi in these leprous patients. Acute leprous reactions frequently occur in the patients seen in Hawaii.

With respect to the whitish globular bodies seen in the iris, there seems little doubt that these are true lepromas. Kuriks obtained a post-mortem specimen of such an eye and was able to show that these structures were indeed miliary lepromas, as had been surmised by Jeanselme and Morax as far back as 1898.

I can also confirm the eccentricity of the leper's reaction to purified protein derivative. A large series of such diagnostic tests have been

done, and a small number of positive reactions have been obtained. In some of my cases negative reactions were obtained even in the presence of both leprosy and tuberculosis, two diseases which are found together with puzzling frequency.

DR. FORREST J. PINKERTON, Honolulu, Hawaii: When one studies the pathologic process in the anterior segment, it would seem that a secondary glaucoma must necessarily follow, and such would certainly be the case if, as Dr. Prendergast pointed out, there were not an early atrophy of the ciliary processes. I have rarely found glaucoma in the many patients I have examined.

I must confirm Dr. Prendergast's report on the absence of changes in the optic nerve, choroid and retina due to leprosy. I have examined the eyegrounds of all the patients coming under my observation, many of them year after year. More than 50 per cent of these patients give positive Wassermann reactions (not necessarily syphilis), hence is it not likely that many of these ocular involvements are due instead to syphilis or to some other causative factor? Also in view of the large number of cases of tuberculosis and of tuberculous complications in leprosy patients, why assume that the lesions in the fundi are due to leprosy? The condition may have been the well known edema and inflammation that are always present during the leprosy allergic attacks associated with urticaria, edema, excruciating pain and mental confusion. I speak of the so-called "acute leprosy fever."

Slit lamp studies of the acute leprosy iris are difficult indeed. There are too many exudates in the anterior chamber to permit detailed study of a nodular iris in the majority of cases.

Many corneas studied with the slit lamp show numerous irregular, nodular grayish masses in the stroma proper, some large enough to project forward and cause an uneven elevation on the anterior corneal surface. These are nodules and will yield large numbers of bacilli when examined under the microscope.

The phenomenon of acute leprosy fever produces in every patient changes that are widespread and involve all parts of the body, the cornea included. The so-called keratitis punctata superficialis is not necessarily a leprosy entity, as leprosy involves the deeper portion of the cornea. Slit lamp examination might correct the reports of some other observers that "keratitis punctata superficialis" is due to leprosy.

There is one lesion of the cornea involving the upper margin that fits the picture of leprosy which Dr. Prendergast describes. I am unable to explain its connection with leprosy, but it is found in a majority of cases of ocular leprosy. I speak of that zone of the upper fifth of the cornea which starts out as a nebulous cloud of "flour dust" particles, superficially located, and gradually become more and more opaque, with an occasional fine blood vessel suggesting a pannus, but is not accompanied by conjunctival inflammation.

No cure for leprosy has been found, but experience in Hawaii in the past ten years clearly demonstrates the importance of early segregation, early hygienic treatment and meticulous attention to diet and physical rest. Race and family vulnerability strongly influence the incidence of the disease.

Leprosy in Hawaii is on the decline, and it is my belief that early diagnosis plus care in a sanatorium explains in a great degree this reduction.

DR. T. J. DIMITRY, New Orleans: Ever since the leper's almost fugitive departure from Europe to the Western hemisphere during colonization and from the Scandinavian countries into the United States a hundred years after, he and his disease have disturbed the Americas.

Louisiana, when yet a colony, essayed to solve the problem of the leper and took its responsibility seriously. On three occasions she established a lazaretto for the lepers' care. Today, she is host to the nation's lepers. Government control has not enlightened but, to the contrary, has succeeded in keeping the riddle stationary and in handicapping the initiative.

My interest in the leper dates back some thirty years, and throughout this period I have noted neglect to study him in his lair, this neglect being less today than when I first became interested. For some unaccountable reason the physician has not availed himself of the social worker in the lepers' care, yet such workers were ready to serve had they been called on.

The leprous lesion of the eye is not unlike that of the skin and mucous membranes of other parts. It is not primary but is a response to the general pathologic condition. In consequence, the ocular lesions are treated symptomatically in the hope that general improvement will result.

Though a leprous nidus in the nasal cavity commonly exists in 90 per cent of the cases in the early stage, the nasal lesion is not necessarily responsible for the ocular leprosy. The progress of the disease is not by a continuity of surfaces. The characteristic active lesions in the eye, the round and epithelial cell infiltration diffusing the meshes of the conjunctiva, advancing into the cornea and producing punctate keratitis, bullae, ulcerations and iritis, give the appearance that the supply of organism finds its way from the nostrils to the eye.

I do not aim to sustain Sticker's dictum that the nose is the primary site of infection in the great majority of those suffering from leprosy or Heiser's observation that a nasal ulcer is the most important symptom and that there is constantly found in the nasal secretions the leper bacilli. The claim I make is that the ocular condition does not actively exist when the nose is free of pathologic involvement, and I wish to stress the necessity of treating the nose as a means of benefiting the eye. I insist that the leper needs the attention of both the otolaryngologist and the ophthalmologist.

The therapeutic agent I prefer in the treatment of the nose, the eye and the general condition is chaulmoogra oil. I drop this oil into the eye and I spray it into the nose in an emulsified state. Surgical treatment of ocular leprosy is unsatisfactory if the leprous condition is active; however, it is most satisfying if the process has abated.

DR. JOHN J. PRENDERGAST, St. Paul: In reply to Dr. Dimitry, I think that ocular leprosy is almost entirely hematogenous and that when it is found in the eye it can also be found in the nose and in practically any other organ in the body. I do not think treatment of the nose would have any effect on leprous ocular lesions. Moreover, I do not believe that chaulmoogra oil has any effect on ocular leprosy. I tried it with three different methods of administration over a period of several months and did not note any change in the ocular lesions.

EXFOLIATION OF THE LENS CAPSULE (GLAUCOMA CAPSULARIS)

FORTY CASES OF EXFOLIATION

RODMAN IRVINE, M.D.

LOS ANGELES

Through the advent of the slit lamp, study of the lens capsule has been facilitated, revealing pathologic changes of scientific and clinical significance not yet fully appreciated or understood. There is still controversy regarding the most fundamental details of the anatomic structure and pathologic appearance of the capsule, a situation challenging laboratory workers and clinical ophthalmologists alike to record their observations with research intent.

The most complete study of the lens capsule is to be found in Vogt's¹ atlas published in 1931. More recent reports of note concerning exfoliation of the lens capsule are those of Holloway and Cowan,² Grzedzielski,³ Sobhy,⁴ Baumgart,⁵ Waite,⁶ Shapira,⁷ Müller,⁸ Hörven⁹ and Garrow.¹⁰ Relatively little is written in English on the subject, and Hörven's article is probably the best summary to date.

From a study of the literature on exfoliation of the lens capsule and of the cases reported here, certain questions requiring consideration are evolved. These are mentioned at the outset in order to stimulate curiosity and suggest relations in the material presented.

First, with regard to the lens capsule:

(a) With pathologic involvement of the lens capsule, is there any related change in the other glassy membranes?

From the Section of Ophthalmology of the Department of Surgery of the University of Southern California School of Medicine.

Read before the Section on Ophthalmology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 18, 1939.

1. Vogt, A.: *Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges*, Berlin, Julius Springer, vol. 2, 1931.

2. Holloway, T. B., and Cowan, A.: *Am. J. Ophth.* **14**:189, 1931.

3. Grzedzielski, J.: *Arch. f. Ophth.* **126**:409, 1931.

4. Sobhy, M.: *Brit. J. Ophth.* **16**:65, 1932.

5. Baumgart, B.: *Boll. d'ocul.* **12**:560, 1933.

6. Waite, J. H.: *Am. J. Ophth.* **17**:254, 1934.

7. Shapira, T. M.: *Am. J. Ophth.* **18**:31, 1935.

8. Müller, H. K.: *Klin. Monatsbl. f. Augenh.* **98**:653, 1937.

9. Hörven, E.: *Brit. J. Ophth.* **21**:625, 1937.

10. Garrow, A.: *Brit. J. Ophth.* **22**:214, 1938.

(b) Is a separation of an intact superficial capsular layer, as occurs in fire cataract, related to Vogt's senile, granular, dandruff-like exfoliation of the same capsular layer?

(c) Is cataract with exfoliation of the lens capsule possibly a heat or radiation cataract?

(d) With what frequency does Vogt's senile exfoliation occur in different parts of the world, and does solar radiation or heat seem to be a factor in the frequency?

(e) Could the particles seen in "senile exfoliation" be a deposition from the iris or the contents of the anterior chamber rather than exfoliation of the lens capsule?

Second, with regard to the lens zonule:

(a) Do the zonule fibers attach themselves to the capsule so as to form a superficial capsular layer, the so-called zonular lamellar layer, and does this layer extend all the way around the lens?

(b) Does pathologic involvement of the most superficial layer of the lens capsule indicate pathologic involvement of the zonule, and, since the zonule is supposedly derived from the tertiary vitreous, pathologic involvement of the vitreous?

(c) If the zonule is affected when there is pathologic involvement of the most superficial capsular layer, does such involvement indicate altered steadfastness of the lens?

(d) In the presence of exfoliation of the zonular lamellar layer of the lens capsule is there an associated change in the permeability of the zonular-capsular diaphragm affecting the exchange of fluid between the chambers of the eye which might result in glaucoma, the so-called diaphragmatic glaucoma of Hörven?

Third, with regard to the immediate clinical significance of exfoliation of the lens capsule:

(a) Is the glaucoma frequently accompanying senile exfoliation of the lens capsule the result of plugging up of the drainage channels of the eye by the exfoliating particles?

(b) If this is true, should treatment of such glaucoma be altered according to its genesis, or, more important, can the onset of glaucoma be prevented in eyes with exfoliation?

(c) Does exfoliation per se alter the prognosis of any concomitant pathologic condition, such as glaucoma or cataract?

The purpose of this paper is to present personal observations as they pertain to some of these problems. These observations include the results of a study of data on 14 cases of glaucoma capsularis taken from the records of the Massachusetts Eye and Ear Infirmary; the results

of examination for exfoliation of the lens capsule of 235 patients with cataract seen consecutively at the Government Ophthalmic Hospital, Madras, India, 18 of whom had such exfoliation, and the results of a study of 8 cases of exfoliation of the lens capsule seen in Los Angeles.

It is necessary at the outset to differentiate senile exfoliation of the lens capsule from other forms that are known to occur, especially the zonular lamellar separation of fire cataract. At present there is no known connection between the two conditions. In 1922 Elschnig¹¹ described in glass blowers separation of the most superficial layers of the lens capsule, a condition known as zonular lamellar separation because of the belief that the so-called zonular layer of the capsule is the part that separated (fig. 1 *A*). The detached membrane is seen as an intact, more or less transparent pellicle, partially separated from the lens, the separating edges curling up in scroll-like fashion or lying free. The break in the membrane probably takes place near the equator, and the axial ends curl up toward the anterior pole. The capsule is usually not detached over the anterior pole. The distinguishing feature of this type of separation is the intactness of the detached membrane. This condition accompanies cataract which develops after ten or fifteen years of exposure to the glass furnaces. Glaucoma does not develop in these patients.

Somewhat similar detachments of the anterior capsule have been described following trauma¹² in cases of hypermature cataracts and occasionally with dislocation of the lens.¹ A woman aged 76 was seen at the Los Angeles County Hospital showing a delicate, transparent, pellicle-like membrane attached to the anterior pole of the lens with the edges curled up as described in zonular lamellar separation. There was nuclear sclerosis, but the fundus could be seen readily. The iris was freely movable but showed the presence of three or four old Koeppe nodules, and one of these was attached to the membrane by a fibrous strand. This membrane was not the type seen as an inflammatory deposition. Its transparency, delicateness and homogeneity pointed more to its being a definite separation of a part of the capsule, probably pulled off in this case by adhesions to the iris. The inflammation of the iris was never marked, and at the time of writing there is no inflammatory activity. The patient does not recall any undue exposure to heat or radiation. She is not a professional cook. This case is cited to show that the lens capsule sometimes undergoes a change that will allow separation of the most superficial layers simulating that seen in fire cataract. Among the cases subsequently described are 2 (cases 13

11. Elschnig: *Klin. Monatsbl. f. Augenh.* **70**:325, 1922.

12. Frenkel, H., and Dejean, C.: *Arch. d'ophth.* **49**:753, 1932. Butler, T. H.: *Am. J. Ophth.* **21**:1032, 1938.

and 14) in which dilatation of the pupil left a faint configuration of the iris markings on the lens capsule, as though the capsule were etched by the iris. The scratches were too fine and delicate to be pigment streaks, and here again was the suggestion of a superficial capsular layer that was so changed as to be ready to rub off. These findings might be a clue as to the beginnings of the exfoliation described by Vogt. In Vogt's atlas similar pictures are shown but are not discussed from this point of view.

HISTORICAL ASPECT

In 1925 Vogt described 12 cases of what he called "exfoliatio superficialis capsulae anterioris." A description of the same condition had been given by the Finnish writer Lindberg¹³ in 1917. Bedell¹⁴ was the first to describe the condition in the American literature. In 1925 he reported 2 cases of "unusual capsular changes," which, from the descrip-

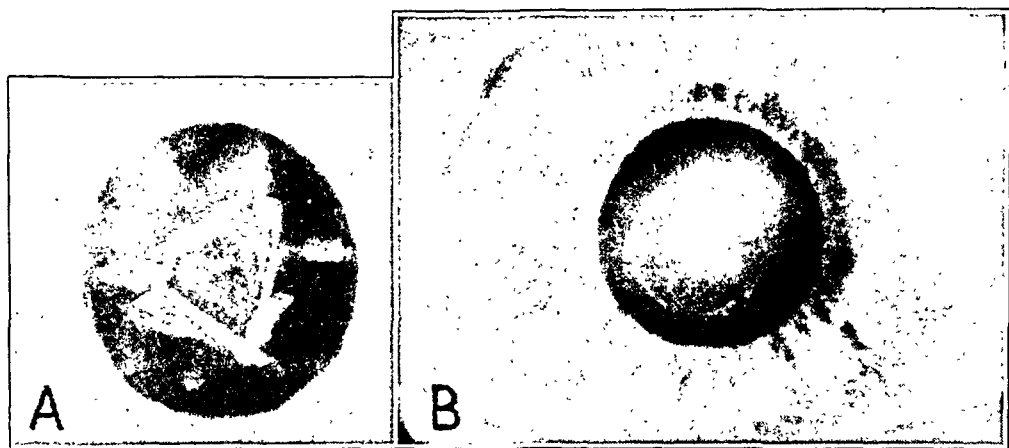


Fig. 1.—*A*, diagrammatic representation of separation of the capsule in glass blower's cataract. *B* (case 22), photograph showing a clearly defined central pupillary disk as seen with the pupil partially dilated.

tion and drawings, are typical cases of exfoliation. Since Vogt's description some forty or fifty papers on the subject have been published, and by 1931, 45 cases had been seen in the Zurich clinic. Vogt and other observers have brought forth clinical and pathologic observations attempting to prove that the anterior layers of the lens capsule come off in small particles or flakes. Only a few observers, notably, Handmann¹⁵ and Busacca,¹⁶ have expressed the belief that the particles are a deposition rather than an exfoliation. Since a large percentage of the

13. Lindberg, 1917, cited by Hörven.⁹

14. Bedell, A., in *Contributions to Ophthalmic Science, Dedicated to Dr. Edward Jackson in Honor of His Seventieth Birthday*, Menasha, Wis., George Banta Publishing Company, 1926, p. 148.

15. Handmann, M.: *Klin. Monatsbl. f. Augenh.* **76**:482, 1926.

16. Busacca, A.: *Arch. f. Ophth.* **119**:135, 1927.

patients showing this condition have glaucoma, which apparently comes on after the exfoliation, Vogt suggested for them the classification, "glaucoma capsularis." Clinically, this condition is distinct from the separation of the capsule seen in fire cataract and should have separate classification even though the same region of the same capsular layer is involved.

DESCRIPTION OF EXFOLIATION

The appearance of the exfoliating material can best be compared to dandruff being rubbed off the lens by the play of the iris (case 19 and fig. 2 *A*). The dandruff-like particles are seen on the edge of the iris as a pale bluish fluff against the atrophic border of the iris (case 24 and fig. 2 *B*). In cases of severe involvement there is a gross sheetlike separation coincident with these fine particles (case 26 and fig. 2 *C*). The separation takes place at the polar end, but the granular dandruffy appearance is always present, being visible on the sheets that are curling up. It is as if a deeper layer were exfoliating in sheets while the more superficial layer was coming off in flakes (case 26 and fig. 2 *D*). The picture reminds one of skin exfoliating after sunburn, when the dead skin may come off as sheets or in small particles, depending on the acuteness of the process and the amount of rubbing to which the sunburned surface is subjected. Analogous factors might explain the difference in the types of separation of the same part of the lens capsule in fire cataract and in senile exfoliation. However, as yet there is no evidence that heat and radiation are etiologic factors common to these two conditions.

Vogt noted the following triad as constituting the syndrome of the senile type of exfoliation. The first is the presence of a central grayish disk in the pupillary space (case 22 and fig. 1 *B*). The edges of this disk may or may not be separating and curled up. One gets the impression that this disk is capsule ready to exfoliate but that it has not done so because the iris has not rubbed over it. The disk is usually the size of the minimal pupillary space. Vogt has been able to reduce its size by the use of miotics over a considerable period of time. Wollenberg¹⁷ reported a case in which there were an immobile pupil in one eye and no exfoliation and a normal pupil in the other eye with exfoliation. The disk is not found in about 10 per cent of the patients, suggesting that the entire anterior capsular surface is not equally affected and rubbed off by the iris, as Vogt assumed. It may be that the capsule is more adherent at the anterior pole or, for some reason, is not as much affected by the etiologic agent as is the peripheral part. There are no reports of exfoliation having been observed on the posterior capsule.

17. Wollenberg, A.: *Klin. Monatsbl. f. Augenh.* **77**:128, 1926.

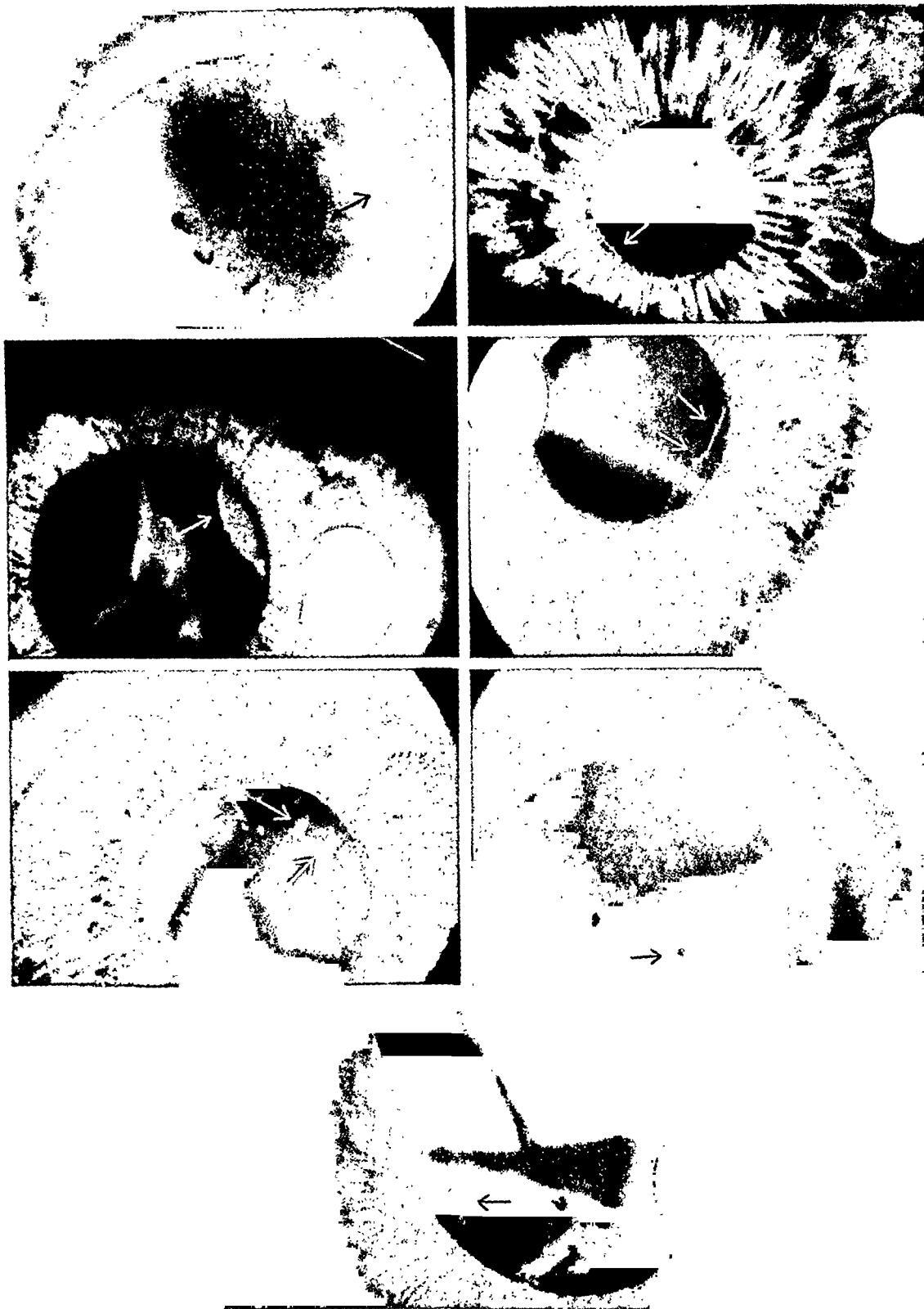


Fig. 2.—Colored photographs of types of exfoliation of the lens capsule described in the text: *A* (case 19), granular, scurflike appearance of exfoliating capsule seen near the edge of the iris; *B* (case 24), dandruff-like particles of the lens capsule on the edge of the iris in the lower left corner; *C* (case 26), gross sheetlike separation of the capsule seen curling back toward the edge of iris in the right upper area of the pupillary space; *D* (case 26), exfoliation in the lower right corner, where a second layer of exfoliation is faintly discernible; *E* (case 25), faint bridgelike structure of exfoliating capsule connecting the pupillary disk with the peripheral band; *F* (case 19), piece of exfoliating capsule photographed on the posterior surface of the cornea (the blurred appearance of the iris and the lens) is due to the fact that the focus was set on the cornea, and *G* (case 19), exfoliation of the lens capsule on the capsular remnant.

The second member of the triad is the peripheral band of exfoliating capsule, usually separated from the central disk by a band of what appears to be clear, normal capsule (case 25 and fig. 2 *E*). Often one or more bridges of granular structure stretch from the disk to the peripheral band. The width of the band varies considerably. It is more often in the outer third of the distance from the anterior pole to the equator, but it may be nearer the center or on the periphery. It is marked by granular radial figurations, which Vogt and Busacca attributed to the configuration of the posterior surface of the iris. The axial border is usually turned up and frayed. The equatorial boundary may, according to Hörven, merge into capsule of normal appearance, or it may be clearly visible, having radial granular projections which are higher than those on the axial border and which, according to Vogt, may have some relation to the attachment of the suspensory ligament. Separation of the capsule at the equatorial border has never been observed.

The third member of the triad is the deposition of fluffy exfoliated particles on the edge of the iris (case 24 and fig. 2 *B*). Hörven stated that these are never seen on the anterior surface of the iris but accumulate along the edge as little masses of fluffy, scurflike flakes. In cases 5 and 17 reported in this paper, what appeared to be exfoliated capsular particles were seen on the stroma of the iris. The presence of released pigment and atrophy of the iris, especially the edge, are often associated with exfoliation. Whether or not these degenerative processes are more extensive than would be found in a similar age group remains to be determined.

Vogt stated that the only member of the triad invariably present is the peripheral band. Hörven added a fourth finding which he considered equally constant, namely, the presence of these scurflike flakes on the fibers of the suspensory ligament. This observation warrants careful consideration, as it might be indicative of zonular pathologic involvement. Hörven has examined 150 eyes with chronic simple glaucoma through an operative coloboma of the iris which exposed the edge of the lens, and in 128 of these he saw typical exfoliation. He did not mention the suspensory ligament specifically in these eyes, but he stated that in eyes in which he had seen the suspensory ligament, including excised eyes, he had seen these exfoliating flakes in all. There is no report in the literature by any one with clinical experience comparable to his in this regard.

Vogt claimed that the appearance of exfoliation changes from time to time, depending on the activity of the pupil. In some cases one can almost follow the transference of the exfoliating flakes onto the edge of the iris, as in case 5. In this case and in case 17, as noted previously, these particles were seen on the stroma of the iris as well as on the edge.

In case 9 exfoliating particles were discernible in the anterior chamber. I have also noted and photographed the flakes on the posterior surface of the cornea (case 19 and fig. 2 *F*). Before I presented these cases at the meeting of the California Medical Association in May 1938, as far as I know the clinical observation of particles in these locations had not been recorded. Since then Garrow¹⁰ has reported having seen the exfoliated particles on the stroma of the iris. Barkan¹⁸ mentioned seeing capsular shreds on Descemet's membrane and in the iridocorneal angle on gonioscopic examination in cases of glaucoma capsularis. I have seen no other reports of gonioscopic findings.

MICROSCOPIC APPEARANCE

Vogt, Sobhy and Busacca have shown pathologic sections in which hyaline particles are seen on the lens capsule, the posterior surface of the iris, and in the iridocorneal angle. Vogt has also shown the superior layers of the capsule to be lamellated and irregular in width, which he assumed was due to exfoliation of the capsule in parts. The pathologic demonstrations of the condition are not as convincing as the slit lamp picture. It seems strange that by slit lamp examination the hyaline particles have such a frosted appearance compared to the relatively transparent separating pellicle seen in fire cataract. Perhaps the difference is due to some refractive phenomena related to the multiplicity of particles seen.

METHOD OF EXAMINATION

From the foregoing diagnostic criteria, it is evident that the condition cannot be excluded until the pupil has been fully dilated, exposing the periphery of the lens. When mature cataract is present, exfoliation is difficult to see against the white background, and often in these cases the condition is not readily diagnosed unless there are typical dandruff-like particles on the pupillary border.

Dilatation of the pupil may turn in the edges of the margin of the iris so that the latter, studded with exfoliated particles, cannot be seen. If a mature cataract is present, evidence of exfoliation may be lacking unless the pupillary edge was examined before dilatation (case 9). This important observation has not heretofore been noted in other reports and must be taken into consideration in diagnosing the condition. When examining patients in Madras, routinely, for exfoliation, I soon realized the need of slit lamp examination both before and after dilatation, and even after this precaution was taken, many cases of mature cataract in which there were no dandruff-like particles on the edge of the iris were undoubtedly overlooked. Certainly diagnosis is not made in many cases

18. Barkan, O.: *Am. J. Ophth.* **19**:951, 1936.

in which it would be obvious if a coloboma were present, since one frequently encounters irides, especially in dark races, that dilate with difficulty. The routine method for dilating was instillation of 2 per cent solution of homatropine hydrobromide plus the placing of a pack of epinephrine hydrochloride into the upper and lower conjunctival sacs. If the previously discussed diagnostic criteria are kept in mind and all patients over 50 years of age, including those with glaucoma, are examined both before dilatation and with fully dilated pupils, the incidence of exfoliation reported will be a correct estimation of the true frequency and will contribute more toward an understanding of the condition.

TABLE 1.—*Frequency of the Occurrence of Exfoliation of the Lens Capsule*

Author	Date	Location	Number of Patients Examined	Age	Percentage with Exfoliation	Comment
Rehsteiner: Klin. Monatsbl. f. Augenh. S2: 21, 1929	1929	Zurich	238	Over 60	2	Patients from old-age home; pupils dilated in 148 cases
Trantas ¹⁹	1929	Athens	237	Over 55	15	All capsular degenerative change considered exfoliation; pupil not dilated in most instances
Baumgart ⁵	1933	Bologna	611	Over 50	8	Hospital patients, 50 with glaucoma; of these, 49% with exfoliation; pupils dilated in some cases
Hörven ⁶	1937	Oslo	154	Elderly	12	69 from old-age home; 30 hospital patients without cataract or glaucoma; 35 patients with cataract; all pupils dilated
Irvine.....	1937	Madras	235	Elderly	8	Patients had cataract; all pupils dilated with 2% homatropine and packs of epinephrine
	1938	Los Angeles	276	Over 50	3	All pupils dilated with 2% euphthalmine; 250 cases of cataract and 26 cases of glaucoma

INCIDENCE

As yet, the frequency of Vogt's senile exfoliation is unknown, since the available reports are controversial. It is generally agreed to be a disease of elderly persons, usually with opacities of the lens. The youngest patient on record is a woman aged 45, a patient of Trantas.¹⁹ Two patients with clear lenses, not included in the present series, have come under my observation.

Table 1 compares reports by various authors as to frequency of occurrence of exfoliation of the lens capsule.

19. Trantas: Arch. d'opht. 46:482, 1929.

ASSOCIATION OF EXFOLIATION WITH GLAUCOMA

Most observers have been impressed by the association of exfoliation and chronic simple glaucoma. In 1917 Lindberg¹³ described pupillary flakes in 30 of 60 patients with glaucoma but offered no explanation for the association. Vogt observed cases of exfoliation in which glaucoma subsequently developed; and so he became more and more enthusiastic in explaining the glaucoma as a consequence of the exfoliation. Trantas¹⁹ reported a contradictory case in which there was glaucoma and no exfoliation in one eye, while exfoliation without glaucoma was found in the other eye. In none of the cases reported in this paper was this inconsistency found. At the same time, however, Trantas reported coincident glaucoma in 14 of 42 patients in whom he saw

TABLE 2.—Percentage of Patients with Exfoliation Having Glaucoma

Author	Year	Number with Exfoliation	Percentage with Glaucoma
Vogt ¹	1931	45	75
Busacca ¹⁶	1927	30	90
Trantas ¹⁹	1929	42	33
Rehsteiner: Klin. Monatsbl. f. Augenh. 82: 21, 1929.....	1929	78 (from the literature)	64
Grzedzielski ³	1931	156 (from the literature)	58
Baumgart ⁵	1933	46	63
Sobhy ⁴	1932	24	54
Alling: Arch. Ophth. 56: 1, 1927.....	1927	7	14
Kirby: Arch. Ophth. 4: 93 (July) 1930.....	1930	7	29
Holloway and Cowan ²	1931	3	67
Garrow ¹⁰	1938	10	80
Irvine.....	India	18	44
Irvine.....	Los Angeles	8	50

exfoliation. Table 2 shows the frequency of glaucoma in the presence of exfoliation as cited by various authors.

The next point for analysis in trying to ascertain something more definite regarding the relation of exfoliation to chronic simple glaucoma would be the number of patients with chronic simple glaucoma who show exfoliation of the lens capsule. Statistics on this relation are presented in table 3.

COMMENTARY ON STATISTICS

From the statistics given in table 1 the average incidence of exfoliation in the population over 50 years of age is difficult to state. Vogt concluded that it is about 2 per cent; Baumgart, 8 per cent and Hörven, 12 per cent. The Los Angeles statistics would indicate that it is about 3 per cent. Although the last-mentioned incidence is low in the table, it is probably high for this community, since only patients with cataract

and glaucoma were examined. The high incidence, 8 per cent in India, is probably explained by the fact that here too only patients with cataract were examined, but the cataracts were much more mature than those seen in this country. The incidence in these different parts of the world as yet has not been shown to have any etiologic significance. The search for exfoliation in India was started with the idea that the excess solar radiation and heat of the tropics might be etiologic factors and that exfoliation, like separation of the capsule in fire cataract, might be associated with radiation cataract. Wright²⁰ made a survey of the incidence of cataract in moist and dry regions of India, feeling that cataract in India might be related to infra-red radiation, which in turn would be

TABLE 3.—*Percentage of Patients with Chronic Simple Glaucoma Having Exfoliation*

Author	Date	Number with Glaucoma	Percentage with Exfoliation	Comment
Lindberg ¹²	1917	60	50	Pupils not dilated
Vogt ¹	1931	150	9	
Mulling: <i>Acta ophth.</i> 1:97, 1923..	1923	81	40	
Baumgart ¹⁵	1933	59	49	Pupils dilated
Hörven ⁹	1937	150 (operated on)	85	Coloboma present
		43 (not operated on)	93	Pupils dilated
Garrow ¹⁶	1935	51	16	Pupils not systematically dilated
Irvine	India	33	24	Pupils dilated (see text)
Irvine	Los Angeles	125	4	Pupils dilated in only 26 cases; percentage therefore low

affected by moisture in the atmosphere. There was no appreciable difference found in the incidence in the two regions. The experience in examining patients with cataract in India taught how easily the diagnosis of exfoliation could be missed unless one's attention were focused on this condition and unless the aforementioned procedure for examination were followed, as in case 9. Probably the selection of cases and the degree of thoroughness of examination explain the differences in frequency reported in the literature rather than any real difference in incidence with respect to location. It is interesting that India and Sweden, with extremes of climate, report the highest incidences.

In most cases of exfoliation of the lens capsule there is a coincident cataractous change in the lens. This is to be expected in persons over

20. Wright, R. E.: *Indian J. M. Research* 24:917, 1937.

50 years of age, and no attempt has been made to correlate an exact relation to cataract, as has been done for glaucoma. The present study indicates an increase with age and the extent of cataractous change.

Statistics as to the incidence of glaucoma in cases of exfoliation are more definite and less contradictory than those relating to absolute frequency. Most observers find chronic simple glaucoma in about one-half the patients showing exfoliation. This is seen in table 2.

The data in table 3, namely, the percentage of persons with chronic simple glaucoma showing exfoliation, varies within extreme limits, from about 9 per cent, as stated by Vogt¹ and Blaickner,²¹ to 93 per cent, reported by Hörven.⁹ Undoubtedly the reported low percentages, as in the cases seen in Los Angeles, are too low, and future examinations of eyes with fully dilated pupils will show a higher percentage of exfoliation in cases of chronic simple glaucoma. The 93 per cent reported by Hörven is probably explained on the basis of selection of cases called chronic simple glaucoma. The Indian series showed about 24 per cent of persons with chronic simple glaucoma having exfoliation. A routine examination was not made of all patients with chronic simple glaucoma seen in the hospital at Madras, and those used for the series were those with glaucoma who were found among the patients with cataract. For the most part a diagnosis of glaucoma was not made in these cases until exfoliation was found. Therefore, the incidence of exfoliation in cases of chronic glaucoma in this group is high. The available data as yet do not justify Hörven's belief that chronic simple glaucoma is due to exfoliation of the lens capsule. However, the common association of the two conditions warrants further study as to a cause and effect relation. Conclusions from such a study would undoubtedly affect the treatment in these cases.

TREATMENT

On the assumption that the exfoliating particles plug up the drainage channels of the eye, early intracapsular extraction of the lens has been advised to eliminate further accumulation of these particles. Likewise, when glaucoma is present, extraction of the lens has been advised in place of a decompression operation, even when the lens is relatively clear. In fact, the question arises as to whether or not a relatively clear lens in a patient without glaucoma should be removed in order to prevent future development of glaucoma. On the assumption that the glaucoma instead of being caused by obstruction of the drainage channels is the result of altered permeability of the zonular-capsular diaphragm, probably the same consideration for treatment and prevention arise.

21. Blaickner, J.: *Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch.* **49**:325, 1932.

In an attempt to throw some light on the best methods for treatment of glaucoma in these patients with exfoliation, the records of 14 patients seen at the Massachusetts Eye and Ear Infirmary and 4 seen in Los Angeles were reviewed. These patients were followed for more than six months unless otherwise noted. The results in these cases are summarized as follows: Trephining was done in 6 cases, with control of the glaucoma. It was done twice in 1 case, but the glaucoma was controlled only after subsequent cataract extraction. In another case in which trephining was done the tension was not controlled subsequently. Iridotomy was done in 1 case with control of the tension, but in another case the tension was not controlled. Cataract extraction was done in 4 cases with control of tension. (Three of the operations were intracapsular and one was extracapsular. One of the patients subjected to intracapsular extraction and the patient subjected to the extracapsular procedure were followed for only two months.) Cataract extraction (extracapsular) was done in 2 cases, and the tension was not controlled. In 2 cases tension was controlled by miotics alone for six months and four years, respectively.

This series is too brief and incompletely followed to signify anything more than that the usual procedures for glaucoma can control tension in some cases of glaucoma capsularis.

It is interesting that 4 of the patients in Los Angeles have been known to have had exfoliation for one, two, two and three years, respectively, without glaucoma developing. There is no doubt but that some patients may have exfoliation for a number of years without any symptoms of glaucoma and that once it has developed, tension may in some instances be controlled with miotics for as long as four years. Müller⁸ suggested in cases of exfoliation immobilization of the iris with mydriatics or miotics to prevent rubbing off of capsular fragments that might plug up the drainage channels.

On the basis that the foregoing exfoliation theory of simple glaucoma has some truth in it, it would seem conservative to place all patients showing the condition on miotics regardless of whether glaucoma is present or not. There is probably no justification for removing a relatively clear lens from a person with exfoliation and glaucoma until the usual means of controlling the tension have been tried. Certainly this is true where the life expectancy is not great. Further studies of patients followed longer on different types of treatment may prove that in a relatively young person it is better to remove the lens in capsule as soon as exfoliation is noted. With the present knowledge, other considerations being equal, one is probably justified in removal of a lens with an exfoliating capsule earlier than might otherwise be done. A broad basal iridectomy would seem advisable as a part of the operation.

Exfoliation of capsular remnants was found in 2 instances in this series in which extracapsular extraction was done. In 1 (case 19 and fig. 2 G), extracapsular extraction was done eight years ago, and in the other. (case 11), four years ago. In both there are typical dandruff-like particles on the edge of the iris, and the capsular remnants show frosting and obvious peeling. The exfoliation in 1 of these patients is known to have been present for three years. Neither of them shows any evidence of ever having had glaucoma. It is interesting to speculate that the exfoliation may have occurred after the cataract extraction. Here again the possibility of a long latent period of exfoliation without glaucoma is emphasized.

PROGNOSIS

Concerning the operative prognosis in cases of glaucoma and cataract with exfoliation as compared with that in similar cases without exfoliation, one gets the impression from the literature and from personal experience that it is less favorable in the former. However, no worthwhile series has been presented for comparison of the final outcome for these two groups. The theoretic question of the zonula being affected and the capsule being altered warrants attention when operation is attempted. In cases 3 and 25 the zonula was unusually weak, as the lens dislocated when an attempt was made to grasp the capsule below for a tumbling operation. Vitreous came up over the anterior surface of the lens before any pressure had been applied, necessitating scooping of the lens, with loss of vitreous in both instances. In these cases there was negative pressure in the posterior segment after the section. Of 4 other cases in which the Verhoeff method of cataract extraction was used, there were no complications in 2 (case 24 and 1 in which operation was done at the Massachusetts Eye and Ear Infirmary) and the capsules ruptured in 2 (in both of which operation was done at the Massachusetts Eye and Ear Infirmary). Sobhy reported 3 cases in his series in which dislocation of the lens occurred. In cases 7 and 22 of my series there was a tremulous lens. I am aware of many other cases of exfoliation in which operation was done without complications, but the foregoing experiences are cited to call attention to the possibility of weak zonules, fluid vitreous and friable capsules. In contrast to the foregoing cases, the lens was dislocated with difficulty in case 5 owing to anterior peripheral synechiae, as judged by the presence of iris pigment on the capsule in the region of the equator.

SUMMARY

Senile exfoliation of the lens capsule is more prevalent than is generally recognized. It was found in 8 per cent of 235 patients with cataract examined for exfoliation at the Government Ophthalmic Hos-

pital in Madras, India, and in approximately 3 per cent of patients with cataract seen in Los Angeles. The relation of senile exfoliation to chronic simple glaucoma is more significant than is generally believed. Fifty to 75 per cent of eyes seen with exfoliation of the lens capsule have chronic simple glaucoma. There are insufficient data available for any statement as to the incidence of exfoliation in cases of chronic simple glaucoma, estimations varying from 5 to 93 per cent. The theoretic considerations regarding the causation of glaucoma following exfoliation suggest certain modifications in the care of these patients worthy of attention. Clinical experience gives the impression that the operative prognosis is not as favorable in patients with exfoliation as in a similar group without exfoliation.

SYNOPSIS OF CASES

The patients in cases 1 through 18 were seen at the Government Ophthalmic Hospital in Madras, India. The age recorded for them is often grossly inaccurate. All were examined with the slit lamp both before and after dilation of the pupils with a 2 per cent solution of homatropine hydrobromide and a pack of epinephrine hydrochloride unless otherwise noted. The patients in cases 19 through 26 were seen in Los Angeles. A 2 per cent solution of euphthalmine was used for dilation. This does not give as complete dilatation as was obtained in the first group.

CASE 1.—A woman, aged 70, had a mature cataract in the right eye. When she was examined the capsule was peeling off on the temporal side of the lens in the peripheral zone; there were no dandruff-like particles on the edge of the iris. The tension was normal to touch, and the projection of light was good.

A corneal opacity in the left eye prevented examination of the lens. Absolute glaucoma was present.

CASE 2.—A man, aged 55, had total sclerosis of the lens of the right eye. The capsule appeared thin; there was no peeling or exfoliation seen on the capsule, and there were no dandruff-like particles on the edge of the iris. The tension was normal to the touch, and the projection of light was good.

The left eye contained an immature cataract with a relatively clear anterior cortex and nuclear sclerosis. Exfoliation of the capsule was seen in the extreme periphery of the lens. There were no dandruff-like particles on the edge of the iris; the tension was normal to the touch, and the projection of light was good.

CASE 3.—A man, aged 60, had total sclerosis of each lens. There were dandruff-like particles on the edge of each iris and faint peeling of the capsule at the extreme periphery. The tension was normal to touch, and the projection of light was good. Cataract extraction in the left eye resulted in dislocation of the lens while the capsule was being grasped, necessitating scooping of the lens, with loss of vitreous.

CASE 4.—A man, aged 70, had a hypermature cataract with a folded capsule below in the right eye. There were a few fine dandruff-like flecks on the posterior

aspect of the border of the iris. With the pupil dilated, one or two curled peelings were discernible on the lens capsule on the extreme nasal side. It was difficult to see them because of the white background. The tension was normal to touch, and the projection of light was good.

The left eye showed an immature cataract with relatively clear anterior cortex. The dandruff on the edge of the iris was so marked as to be visible grossly. In the temporal peripheral zone the frosted appearance of the capsule could be seen in radial arrangements, giving a sunflower effect. There was no curling up of the edges. Tension was normal to touch, and the projection of light was good.

It is interesting in this case that dilatation caused folding in of the border of the iris, so that the gross dandruff-like particles on the edge were no longer visible.

CASE 5.—A man, aged 75, was found to have a clear anterior cortex with nuclear sclerosis in the lens of the right eye. Occasional dandruff-like flecks were seen on the pupillary edge. The lens capsule had a frosted appearance above and slightly curled edges. The tension was normal to touch, and the projection of light was good.

In the left eye there were fine subcapsular opacities and anterior cortical spokes. The pupillary border was covered with bluish white knobs or balls of fluff between which small flecks were interspersed. This change was visible grossly. A few of the flecks occasionally were discernible on the stroma of the iris and in the crypts. The lens capsule in the entire peripheral zone presented a frosted appearance with curled up edges. Two or three distinct layers of exfoliation were discernible in places. The frosted appearance of the lens was more globular than usual, and a path of transference of these globule-like particles from the lens to the iris seemed obvious. In some places capsular peeling was seen with a free edge in contact with the iris. The tension was normal to touch, and the projection of light was good.

Intracapsular cataract extraction was done. The lens dislocated with difficulty, as there were anterior synechiae present, leaving iris pigment on the lens. After removal of the lens a remnant of exfoliating membrane was visible on the lower edge of the iris.

The statement as to observation of exfoliated flecks on the stroma of the iris and in the crypts is a direct quotation from notes written at the time of the examination. I was unaware at that time of the statement by Hörven that exfoliated particles are never seen on the stroma of the iris. Had I known this, I would have described the position of these particles more exactly. Similarly, a piece of the exfoliating membrane was seen on the face of the iris in case 19.

The note made by Col. Robert E. Wright on examination of the left eye reads as follows: "Plenty of fluff; balls of fluff on the pupillary edge; lens capsule in a condition of friability as though it would break up easily; green crystals in the lens substance under the capsule; portion of fluff on the iris might be tracked down to joining the lens capsule; in places, two layers of exfoliation noted."

CASE 6.—A man, aged 57, had a mature cataract with a sectored flaky cortex in the right eye. There was a slight amount of dandruff-like flecks on the atrophic edge of the iris; after dilatation this could not be seen. The capsule had a definite frosted appearance with curled edges in the periphery of the lens. The tension was normal to touch, and the projection of light was good.

The left eye contained a hypermature cataract with a folded capsule. No exfoliation was seen on the pupillary edge or on the capsule. The tension was normal to touch, and the projection of light was good.

CASE 7.—A woman, aged 55, had an immature posterior cortical cataract with a clear anterior cortex in the right eye. There were definite dandruff-like particles on the edge of the iris, giving the appearance of felt. The capsule seemed to be coming off in sheets as two distinct layers.

In the left eye there was a hypermature cataract with a tremulous lens. Dandruff-like felt was seen on the edge of the iris. This was more marked than in the right eye. No exfoliation of the capsule was seen against the white background. The patient was treated for chronic simple glaucoma of each eye.

CASE 8.—A man, aged 60, had a mature cataract with a flaky cortex in the right eye. The iris had an atrophic edge, and no exfoliating particles were seen on the edge of the iris or on the lens capsule. The tension was normal to touch, and the projection of light was good.

The left eye contained hypermature cataract with a folded capsule. Typically definite dandruff-like particles were seen on the edge of the iris without there being discernible against the cataractous background any exfoliating particles on the capsule. The tension was normal to touch, and the projection of light was good.

CASE 9.—A man, aged 70, had an immature cataract with a relatively clear cortex in the right eye. No exfoliation of the lens capsule or particles on the iris were seen. The tension was normal to touch, and the projection of light was good.

There was a morgagnian cataract in the left eye. The pupillary edge was covered with exfoliated particles, and some particles were discernible in the anterior chamber. The pupillary border was slightly atrophic. The capsule seemed to be definitely exfoliating on one side with slight curling of the edges. There was peeling on the other side of the capsule in gross flakes. The tension was normal to touch, and the projection of light was good.

The exfoliation of the lens capsule was difficult to see in this case because of the mature cataract. Were it not for the marked dandruff-like particles on the pupillary edge and the few gross flakes seen on one side of the lens, the fine stippling of the capsule seen on careful focal illumination would have been overlooked.

CASE 10.—A man, aged 58, had an immature cataract in the right eye. Definite dandruff-like particles of exfoliation were seen on the edge of the iris. The pupil was not dilated. The tension was markedly increased. The condition of the left eye was the same as that of the right. There was glaucoma in each eye.

CASE 11.—A man, aged 60, had a mature cataract with a flaky anterior cortex in the right eye. No exfoliation was seen on the lens capsule or on the edge of the iris. The tension was normal to touch, and the projection of light was good.

Cataract extraction had been done on the left eye four years before. The edge of the iris was atrophic, but no dandruff-like flecks were seen. After dilatation anterior capsular remnants showed definite exfoliation characterized by frosting, stippling and slightly curled edges of the separating part. There was no evidence of cupping of disk. The tension was normal to touch, and the projection of light was good.

CASE 12.—A man, aged 65, had an immature cataract with a relatively clear anterior cortex in the right eye. There were definite dandruff-like flecks on the edge of the iris and some scaling of the lens capsule. The left eye was practically the same as the right. The tension was elevated in each eye, and there was definite cupping of disks. Glaucoma was present.

CASE 13.—A man, aged 60, had a mature cataract with a sectorized cortex in the right eye. The edge of the iris was atrophic, and no exfoliation was seen. After maximum dilatation, a few faint scratches were visible on the lens capsule but no exfoliation. The tension was normal to touch, and the projection of light was good.

The left eye contained a mature cataract. Definite dandruff-like particles were present on the pupillary edge and there were a few shreds of exfoliated material in the periphery of the lens. The mature cataract made it difficult to see them. The tension was normal to touch, and the projection of light was good.

CASE 14.—A man, aged 60, had a hypopyon ulcer in the right eye. It was impossible to examine the lens. Glaucoma was present.

There was an immature cataract with a relatively clear anterior cortex in the left eye. Definite dandruff-like particles were seen on the atrophic edge of the iris. There were pigment deposits on the lens suggesting previous slight synechiae. After dilatation, no definite exfoliation as characterized by frost or stippling of the lens capsule was seen, but instead there were noted fine radial scratches, vaguely suggesting exfoliation. Some fine pigment granules were seen interspersed between these scratches. The pupil would dilate only about one half, owing to definite peripheral synechiae, so that the peripheral zone of the lens could not be examined. However, the dandruff-like flecks on the edge of the iris were typical. The tension was moderately high.

This case and case 13 are those to which reference was made in the text suggesting that scratches of the lens capsule may be the first visible signs of a change in the superficial layer preceding exfoliation.

CASE 15.—A man, aged 60, had a mature cataract in the left eye. Definite dandruff-like particles were seen on the edge of the iris. The pupil was not dilated. The tension was normal to touch, and the projection of light was good. The right eye was not examined.

CASE 16.—A man, aged 60, had an immature cataract in each eye. Definite dandruff-like flecks were seen on the edge of each iris, and there was fine stippling of the capsule. Glaucoma was present.

CASE 17.—An elderly man had a mature cataract in each eye. Definite dandruff-like flecks were seen on the edge of each iris. The pupils were not dilated. Chronic simple glaucoma was present.

CASE 18.—A man, aged 60, had an immature cataract in each eye. Dandruff-like particles were seen on the edge of each iris. The pupils were not dilated. Glaucoma was present.

CASE 19.—A woman, aged 60, underwent extracapsular cataract extraction on the right eye eight years before the reported examination. At this examination typical exfoliation particles were seen on the capsular remnants. The tension was normal, and there was no cupping of the disk. Vision was 20/20 with correction.

There was mature nuclear sclerosis of the left eye. Peeling was seen on the lens capsule, the exfoliating material coming off in loops, and a large amount of peeling was seen on the edge of the iris. A strand of what appeared to be capsule was seen on the stroma of the iris, and in addition there was a large piece of exfoliating material on the posterior surface of the cornea. The tension was normal, and the projection of light was good.

CASE 20.—A man, aged 76, had an immature cataract in the right eye with marked exfoliation of the lens capsule. No mention was made of dandruff-like flecks on the edge of the iris. The tension was elevated, and the disk was cupped. The eye had been operated on for glaucoma, there being evidence of an iridotomy above and an old nonfiltering trephine opening.

Extracapsular cataract extraction had been done on the left eye with loss of vitreous. There was considerable iritis. No details of exfoliation could be made out. The tension was soft, and vision was hopelessly lost. A more detailed record was not available as the patient was referred from out of town.

After operative manipulation of the iris, I have frequently found that the dandruff-like particles previously present on the edge of the iris have disappeared.

CASE 21.—A man, aged 69, had right anophthalmos. Early lenticular changes were present in the left eye. Trephining had been done with complete iridectomy. Marked exfoliation of the lens capsule and dandruff-like particles on the iris were seen. There was definite glaucomatous cupping of the nerve. The tension was normal to touch, and at the time of writing it has been controlled for over a year. Vision was 20/50.

CASE 22.—A woman, aged 65, had a posterior cortical cataract in the right eye. The lens was tremulous. No dandruff-like flecks were seen on the atrophic edge of the iris. There was typical exfoliation of the lens capsule with curling up of the separating sheets. The central pupillary disk was clearly defined. The optic nerve was normal. Tension was normal.

There was an immature posterior cortical cataract in the left eye. There were moderate exfoliation of the capsule in the periphery, a few keratic precipitates on the posterior surface of the cornea, a slight aqueous flare and a few cells in the anterior chamber. The tension was normal, and the projection of light was poor.

CASE 23.—A woman, aged 70, had questionably slight cupping of the disk of the right eye and a corresponding field defect. No exfoliation was seen. Vision was 20/30.

There was slight bedewing of the corneal epithelium of the left eye. The lens was sufficiently clear to allow good examination of the fundus, which showed glaucomatous atrophy and cupping of the nerve head. There was definite exfoliation of the capsule with a slight amount of dandruff-like particles on the edge of the iris.

CASE 24.—A woman, aged 82, had an immature cataract in the right eye. There were dandruff-like particles on the edge of the iris and the typical appearance of an exfoliating lens capsule with slightly curled edges in places. The tension was normal. No field defects were present. Vision was 20/100. A hazy view of the fundus did not reveal any gross pathologic involvement.

There was a mature cataract in the left eye. Dandruff-like particles were seen on the edge of the iris, and there was a typical exfoliative change of the peripheral capsule. The tension was normal, and the projection of light was good. Iridectomy was done with delivery of the lens in capsule by the Verhoeff method. The lens dislocated readily. The patient has been followed for two years since operation and at the time of writing has vision of 20/20 with correction. There is no evidence of glaucoma in either eye.

CASE 25.—A woman, aged 76, had an immature cataract in the right eye. A slight amount of dandruff-like particles was seen on the edge of the iris. The pupil dilated, revealing marked stippling and exfoliation of the capsule in sheets. The pupillary disk was clearly demarcated on one side. On the other side it formed a bridge continuous with the peripheral zone of the exfoliating capsule. This bridge from the disk to the peripheral band showed extremely fine radial stippling which stopped at the margin of the pupillary disk, merging into the relatively normal-appearing capsule of the disk. The capsule of the pupillary disk appeared definitely different from that of the periphery, both where the peripheral capsule was obviously exfoliating and where it presented a homogeneous gray appearance, like desiccated skin that is ready to peel off but has yet not done so. One got the impression that all the capsule in contact with the iris had been affected while the pupillary disk remained more normal. The tension was normal, and there were no changes in the visual field.

There was a mature cataract in the left eye. A slight amount of dandruff-like particles was seen on the edge of the iris, and there was definite stippling of the capsule in the periphery. The tension was high normal, and there were no changes in the visual field. Cataract extraction was done. There were definite anterior peripheral synechiae. As the capsule was grasped with forceps, the zonule ruptured, allowing fluid vitreous to come up over the lens. The lens was scooped in the capsule with considerable loss of vitreous. The postoperative course was stormy, but six months after operation vision was 20/25 and tension was normal.

CASE 26.—A woman, aged 76, had a nuclear opacity in the right eye. There was marked exfoliation of the lens capsule, the exfoliating material coming off in sheets. Dandruff-like particles were seen on the edge of the iris, and there was slight cupping of the nerve head with a definite field defect. Vision was 20/25.

There was nuclear sclerosis in the left eye with anterior spokes. Marked exfoliation of the capsule was seen which curled up in sheets. There were dandruff-like particles on the edge of the iris and cupping the nerve. A field defect was present. Vision was 20/40. At the time of writing the tension and fields have been controlled for four years with miotics.

CASES 27 to 40 (from the records of the Massachusetts Eye and Ear Infirmary).—Eleven men and 3 women, ranging in ages from 49 to 75, had glaucoma capsularis. They are discussed in the text only from the point of view of treatment. The exfoliation present in these cases was not recorded in detail.

The definition of the exfoliating particles is not as clear on the color plate as on the kodachrome transparencies.

Col. Robert E. Wright gave me permission to examine patients at the Government Ophthalmic Hospital in Madras, India.

Mr. Russell Stimson, of Los Angeles, who is responsible for the ingenious method of photographing the anterior segment, has made the actual photographs of the lens capsule possible for the first time.

ABSTRACT OF DISCUSSION

DR. DANIEL B. KIRBY, New York: The subject dealt with here is one of importance in the fundamental study of the anatomic structure, physiologic function and pathologic picture of the lens capsule. The findings prove that the capsule is laminated to at least the degree that the most superficial lamella is separate from the main body of the glass membrane; that the capsule is not indestructible, and that by one degenerative process at least its physiologic function may be altered and the selective two-way permeability mechanism, which is so necessary for the nutrition, growth and metabolism of the lens, may be changed. One may by inference assume that this function may be disturbed by other degenerative processes. In the cases which I have observed there has been in the early stages no sign of death or change in the epithelium of the lens. The cataracts observed have been mostly of the sclerosed type. I see no reason for any attempt to link exfoliation with any form of radiation cataract. The distribution and incidence do not suggest any connection.

I believe the occurrence of glaucoma in these cases to be a coincidence and dependent not on blockage of the channels of exit of the aqueous from the anterior chamber by the dandruff-like scales which came off the lens capsule but on the involvement of the endothelial cells of the iridocorneal angle by the process of degeneration. That the endothelial cells suffer is evident in the process of dystrophy of the corneal endothelium associated with the development of excrescences or warts on Descemet's membrane, such as Basil Graves described so well. The two conditions, dystrophy of the capsule and of the endothelium, are often associated.

The incidence of glaucoma in 50 or more per cent of cases of exfoliation does not prove that the exfoliation causes the glaucoma but only that the two are associated. The only logical treatment for the glaucoma in these cases is a filtering operation. I should not consider the removal of a relatively clear crystalline lens from an eye with useful vision to be justified on the basis of the control of the glaucoma. Dr. Irvine suggests a wide dilation of the pupil in cases of glaucoma to help in the search for the signs of exfoliation. I believe that this should be accepted with caution even for the purposes of investigation because of the possibility of unwished for effects of crowding the iris into the angle.

I have not found at the time of the extraction any evidence of excess fragility of the main body of the lens capsule.

DR. PARKER HEATH, Detroit: Dr. Irvine has given a review with comments on published reports and the currently accepted biomicroscopic findings. He has presented a statistical study of the percentage of relations of glaucoma to exfoliation and, conversely, of exfoliation to glaucoma. His illustrations may be said to set a new high in clarity of detail.

I agree with the author that the volume of all published reports is too small and the methods of examination too variable to permit final conclusions to be drawn. The diagnostic criteria, if followed and amplified as indicated in the future, then combined with volume, statistically will be conclusive.

Dr. Irvine's study is especially welcome and needed, for it shows that exfoliation is more common than is generally suspected. Whether or not the management of lenticular changes and glaucoma will vary with the finding of exfoliation of the capsule can be answered only after sufficient bulk of experience and increased knowledge.

The future will have to tell the answers to many questions. Among these are: Does the glaucomatous aqueous alter lens metabolism to produce changes in the anterior capsule? Does the glaucoma precede the changes in the capsule? Can desquamation cause glaucoma? If so, do the free flakes lower resorption quantitatively or qualitatively at the angle capillary or the surface of the iris? Do they block mechanically in the zonule or at the border of the pupil?

The escape of the posterior lens capsule is difficult to reconcile except through participation of the iris or zonule. The effect on the iris of friction, nutritional disturbances and pigmentary reactions also is a moot question.

Is the zonule with the ciliary body, producing mechanical stress or altering the nutrition of the lens anterior to the zonule?

The effects of light and heat over a long period must be considered. Tissue and foreign body reactions to lens flakes would be of interest.

The interesting clinical finding of exfoliation and its relations to the iris, cataract and senescence need disentangling. Such extremes as the notion that all simple compensated glaucoma is due to exfoliation and that of no causal relation must be brought into line. In solving these problems, careful tonometric examination will be important. Biomicroscopic examination will be both a clinical and a research agent. Study of the pathologic picture in the human eye will be supplemented by animal experimentations and studies of dead tissue.

To the practicing ophthalmologist may be assigned a fair share in the solution of the problem. He is continually searching for glaucoma and he is continually caring for patients so afflicted. Accurate observations, careful notes and clinical reports will be his important contribution.

DR. ARTHUR J. BEDELL, Albany, N. Y.: Of my last 26 patients with exfoliation of the lens capsule, only 1 had glaucoma.

Unless the pupil is dilated, the real beauty of the break in the exfoliating ring cannot be appreciated. In my experience the central plaque remains either the same size or gets smaller. The marginal pupillary portion may likewise decrease.

I am sure that if all the ophthalmologists here will become interested in the subject, they will, to their surprise, find that exfoliation is common. I also believe that if they are as careful as Dr. Irvine has been in the tabulation of statistics they will discover that actually an exfoliating capsule should not be called glaucoma capsularis, for too often no glaucoma exists.

DR. T. L. TERRY, Boston: A glaucomatous blind eye giving the typical picture of exfoliation of the lens capsule and "dandruff" on the pupillary margin was enucleated. Microscopic study of sections of this eye showed roughened exfoliating lens capsule. The exfoliation was present not only in the pupillary area but more peripherally under and between the zonular fibers. On the pupillary margin a clump of amorphous acidophilic material was adherent. Its staining properties and its general appearance were similar to the partially exfoliated capsular material. No anterior peripheral synechiae were present. The meshwork of the iridocorneal angle showed considerable sclerosis. A careful study revealed some of the amorphous particulate matter in this region. It is my opinion that exfoliated particles were carried to the iridocorneal angle and produced secondarily the sclerosis of the meshwork and the glaucoma.

In an eye in which glaucoma is prone to develop, only a small amount of foreign material in the iridocorneal angle could be the final exciting factor.

DR. HARRY S. GRADLE, Chicago: To the triad that Dr. Irvine presented, I should like to add a fourth feature, namely, the direct observation of the exfoliated lens capsule material in the iridocorneal angle by gonioscopic methods. It is rather clear and presents a picture that with a little practice can be read easily. At first I thought that it was distinctive in color, but I have found that the color is not a characteristic.

On examining the iridocorneal angle with a magnification of 12 to 15 diameters, one can find the exfoliated lens capsule lying as fuzzy, isolated masses in the iridocorneal angle.

Dr. Sugar, who is working with me at the Illinois Eye and Ear Infirmary on this problem, reports that he has found it in 20 of 250 cases of glaucoma in which he has made the examination. In other words, his figures are about the same as Dr. Irvine's.

This condition is not new. The first cases I saw were in 1923 when Koeppe was here. I was able to show him 2 cases biomicroscopically. In 1 of these cases glaucoma eventually developed, but I have watched the other patient ever since and there has never been any hypertension.

There is more to this subject than appears on the surface. There is no question in my mind that these capsular exfoliations are the cause of glaucoma in a certain percentage of cases, depending entirely on the size of the exfoliated material and on how much of the iridocorneal angle is thereby blocked. When the elimination of aqueous from the iridocorneal angle is reduced to such a point that absorption by the iris cannot carry the load, then characteristic signs of hypertension appear.

All ophthalmologists agree that the difference between primary and secondary glaucoma is merely that the cause of secondary glaucoma is known while the cause of primary glaucoma is unknown. A certain percentage of cases of primary compensated glaucoma are going to have to be reclassified as secondary glaucoma because of the findings of capsular exfoliations as the cause of blocking of the iridocorneal angle.

DR. S. RODMAN IRVINE, Los Angeles: In regard to the association of exfoliation of the lens capsule with glaucoma, the majority of statistics indicate that about 50 per cent of cases of exfoliation are associated with glaucoma. In my series that is a conservative estimate, as a diagnosis

of glaucoma was made only when the disease was obviously present. I did not have time in India to make tonometric studies, and unless there was a definite glaucomatous field defect or definite cupping and the tension was raised above normal on palpation the condition was not called glaucoma. Undoubtedly, then, the number of cases of exfoliation associated with glaucoma are underestimated, and the actual incidence would be much higher than 50 per cent.

The cases seen in this country are probably seen earlier, before glaucoma has developed, and that is why Dr. Bedell has not observed cases of exfoliation with glaucoma. In the report by Dr. Bedell on "Unusual Capsular Pathology," glaucoma was present in 1 of the 2 cases he described.

As to the possibility of a radiation cataract, although I do not feel that this is the etiologic agent, it is noteworthy that exfoliating material is never seen on the posterior lens surface. If the change were of senile or degenerative nature, one might expect to find it on both surfaces of the lens, although again this has not been determined.

Dr. Kirby stated that he does not feel that these lenses have friable capsules, and yet he finds the sclerosed and hard type of cataract. This type of cataract tends to have a more friable capsule. In my hands the capsules seem to break more easily. Perhaps I break them because I anticipate difficulty.

Dr. Gradle's finding of exfoliated material in the iridocorneal angle is interesting. Dr. Barkan has reported this finding too. I have looked for it, but the material is so white that I have had difficulty with my apparatus seeing it against the white background of the angle. For a long time it was felt that the material is never on the stroma of the iris because it is so difficult to see, but I now have pictures definitely showing the material in this location.

A METHOD OF ULTRA CLOSE-UP PHOTOGRAPHY IN OPHTHALMOLOGY

RODMAN IRVINE, M.D.

AND

RUSSELL L. STIMSON

LOS ANGELES

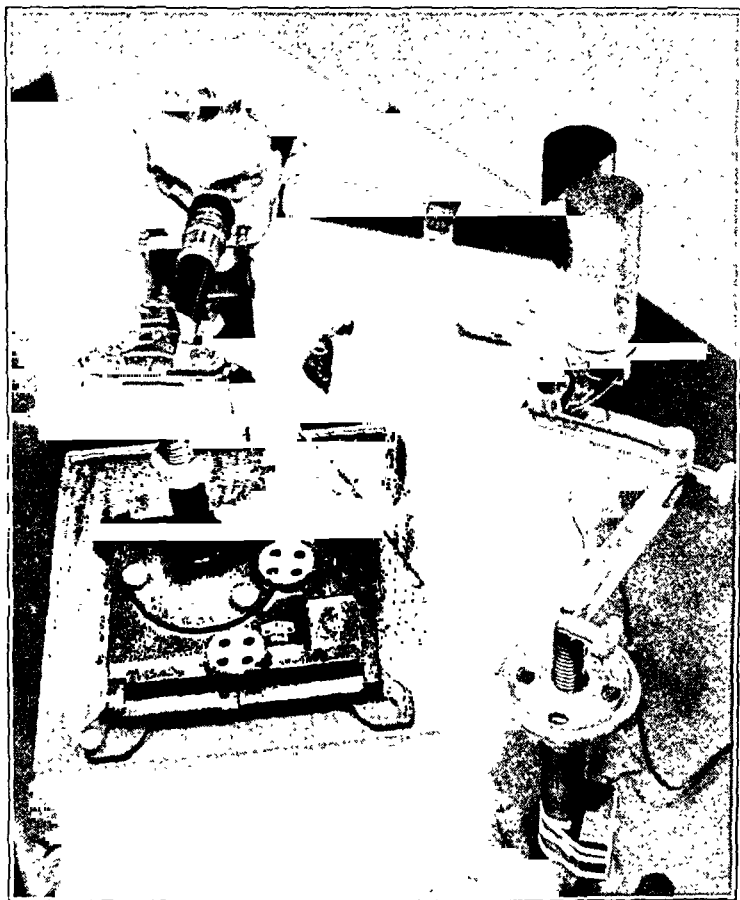
The photography of small detail of the eye has hitherto been difficult and not altogether satisfactory. Best results require magnification of the image on the film at an exposure of not more than one two-hundredth second, and the lens should be stopped down to obtain sufficient focal depth. The high level of illumination necessary for the exposure must be supplied from a flashlamp in order not to be injurious or uncomfortable to the patient. A single lens reflex camera is required to provide an accurate means of focusing.

We have mounted an Exacta camera on a compound base and fitted a slidably adjustable tube behind a 50 mm. Tessar lens. The minimum length of the tube sets the lens 150 mm. from the film plane to obtain a magnification of two times. The maximum length of the tube is 300 mm. to provide a magnification of five times. To facilitate the setting of the lens iris, a handle is fitted to a ring mounted on the adjustment ring, and a movable stop is placed on the front of the lens mount. The iris setting is predetermined, focusing is done at maximum aperture and the handle is rotated to the proper setting just before exposure. A lens hood is necessary to shield the lens from light reflections from the lighting device.

Small detail is often difficult to find in the irregular surface of a ground glass. We have cemented a thin clear strip of glass such as is used in the Leica focusing copy attachment on the ground glass. The millimeter scale engraved on the strip measures the size of the image and assists in the choice of magnifying power. We have found the regular chin and face rest to be too unstable for this work and have substituted a tongue blade which the patient grasps with his teeth to hold the head steady. A small flashlight held by an assistant provides a fixation point for the patient.

An adjustable stand holding the lighting device is placed at the side of the camera table. A pivoted base is mounted on the tip of the adjustable arm. Two lamp houses are fitted on the base in such a manner that either lamp house can be rotated to stop in a centered position at the end of a tube 45 mm. in diameter. An aperture is cut in each

lamp house the same size as the tube. A 100 watt frosted lamp in one lamp house is used for focusing. A coupling is set under this lamp, so that the line current is supplied to it only when it is in position at the end of the tube. The other lamp house contains a No. 20 photo flash foil filament lamp which is fired by the synchronizer built in the camera. This type of lamp gives 22,000 lumen seconds of illumination for a peak of one two-hundredth second. A long cable release for the camera is connected to the base holding the lamp houses. As the lamp houses



Instrument used for photography of the eye.

are rotated, the plunger is compressed by a finger on the metal base and the lamp fires just as it takes position.

The light tube is 30 cm. long and is fitted with a $+16.00$ D. lens at the far end. An image of the lamp is formed about 75 mm. from the end of the tube, which is about 12 mm. in diameter. No diaphragm is necessary for pictures of the lens, iris or large corneal defects. A slit lamp beam can be simulated for photographic purposes by placing a frosted celluloid diaphragm in front of the lamp which has a clear rectangular area in the center to allow free passage of light. The

frosted part should pass about one fiftieth as much light as the clear area, so that the area surrounding the image of the clear area will not be completely underexposed.

Kodachrome film is used routinely for the advantage of color differentiation and a picture without silver grain. Enlarged negatives or prints can be made at 20 to 30 diameters. At a magnification of four times the lens is stopped to $f/10$, which gives approximately 2 mm. depth of focus. The shutter is set at bulb exposure to take the whole energy of the quick firing lamp. A standard table as found in a photographic manual will give the necessary variations in aperture for other magnifications.

Clinical Notes

A PLASTIC TRANSILLUMINATOR

T. L. TERRY, M.D., AND R. D. MATTIS, M.D., BOSTON

A transilluminator in order to be completely satisfactory for use on both the anterior and the posterior segment of the eye should have the following properties: (1) small size; (2) light weight; (3) freedom from heat; (4) ease of sterilization; (5) focal light of variable intensities, and (6) atraumatic construction, i. e., a smooth surface and a design based on the anatomic structure of the region.

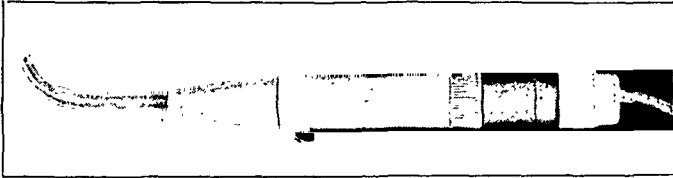


Fig. 1.—Plastic transilluminator.

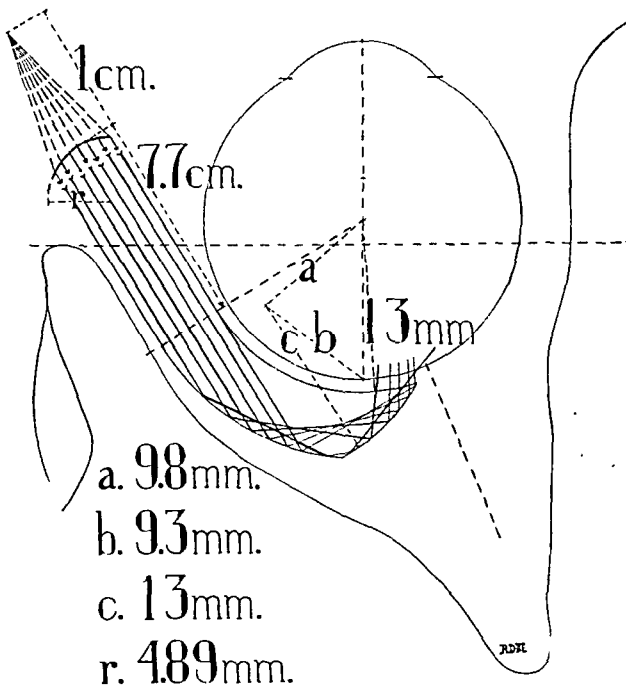


Fig. 2.—Diagram showing physical optics of curved posterior tip.

Finding no such instrument already available, we designed one. It consists of two tips, one curved, of methyl methacrylate (called lucite by du Pont) mounted for focusing before a powerful prefocused bulb in a "penlight" handle and connected to the power supply by a detachable cord (fig. 1).

Read before the Section on Ophthalmology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 18, 1939.

The tips are cylindric, polished rods 5 mm. in diameter. The end of each rod which is placed in the mounting near the light source is ground and polished to a convex collimating surface, thereby refracting the incident rays and directing the resultant parallel rays within the rod. The straight tip, which is used in the conjunctival sac, terminates as a polished plane contacting surface. The curved posterior segment tip was optically designed to reflect the parallel rays internally, so as to form on emergence a beam directed forward and converging with a relative focal distance of about 20 mm. (fig. 2). This beam illuminates the sclera over an elliptic area of approximately 3 by 5 mm.

The lamp housing is suitable for the use of a standard type General Electric PR-2 bulb or of a more powerful bulb on a current of 6 volts which is controlled by a rheostat in order to vary the intensity of the light.

The instrument is small and of light weight and remains cold on prolonged use. Although the handle and cord can be boiled, the tips are sterilized in a solution of mercury bichloride (1:1,000).

In actual use on human beings, adequate transillumination of the normal eye is obtained with a minimum of trauma. The curve of the posterior segment tip is satisfactory for placing the free end into Tenon's capsule and directing the beam through any desired point on the posterior portion of the globe. The straight tip furnishes satisfactory illumination of the anterior segment of the eye.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

VISUAL HALLUCINATIONS AND THEIR NEURO-OPTICAL CORRELATES

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AND

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Visual hallucinations appear so frequently in association with both mental and organic disease of the nervous system that it seems curious they have come to be regarded as possessing considerable localizing importance in the diagnosis of tumors of the brain. Indeed, modern textbooks of neurology state that it is possible to make a differential diagnosis between lesions of the temporal lobe and of the occipital lobe on the character of the visual experiences. Jelliffe and White¹ (1935) declared that formed and crude hallucinations appear in association with lesions of the temporal and of the occipital lobe respectively. Brain² (1933) in his textbook stated that visual hallucinations accompanying lesions of the temporal lobe are more highly organized than those due to lesions of the occipital lobe. Brain and Strauss³ (1934), Wechsler⁴ (1935), Grinker⁵ (1934) and Purves-Stewart⁶ (1937) in their textbooks uniformly stated that the visual experiences associated with lesions of the occipital lobe are confined to flashes of light and color and other so-called simple phenomena, while complex and elaborate images are found associated with lesions of the temporal lobe.

From the Neuro-Surgical Service of the Hospital of the University of Pennsylvania.

1. Jelliffe, S. E., and White, W. A.: *Diseases of the Nervous System*, ed. 6, Philadelphia, Lea & Febiger, 1935.

2. Brain, W. R.: *Diseases of the Nervous System*, London, Oxford University Press, 1933.

3. Brain, W. R., and Strauss, E. B.: *Recent Advances in Neurology*, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1934.

4. Wechsler, I. S.: *A Textbook of Clinical Neurology*, ed. 3, Philadelphia, W. B. Saunders Company, 1935.

5. Grinker, R. R.: *Neurology*, Springfield, Ill., Charles C. Thomas, Publisher, 1934.

6. Purves-Stewart, J.: *The Diagnosis of Nervous Diseases*, ed. 8, London, Edward Arnold & Co., 1937.

Moreover, since one does not find references in these works to the fact that visual hallucinations may occur in association with lesions of the optic nerves, chiasm and retina, the inference is that they are solely central phenomena provoked by cortical irritation. It is implied that there are "centers" in the brain irritation of which will produce only "crude" visual impressions, while other areas exist in which irritation by a tumor or other lesion will evoke complex images of persons, scenes or objects in the fields of vision.

This difference in the character of the hallucinations has been accounted for by stimulation of the visuosensory cortex in the first instance and by stimulation of the visuopsychic area in the second (Hoppe,⁷ 1921). Hughlings Jackson⁸ (1889) stated: "If cells of the nervous arrangements of these centers (visual ideation) become unstable to so marked a degree as occurs in epilepsy, the consequence will be that many nervous arrangements will discharge simultaneously and excessively . . . and a visual hallucination results." Kennedy⁹ (1921) explained the occurrence of hallucinations in association with tumors of the temporal lobe on the basis of the revival of infantile memories stored in the temporal lobe and excited into consciousness by the gross irritation of this lobe.

The foregoing hypotheses elaborate the theory of cerebral localization to the point where complex apperceptive and cognitive processes are considered as represented in discrete areas of the brain if not in a single gyrus. According to these conceptions, the inference may be drawn that neither crude nor complex visual hallucinations may be excited by lesions of the retina, optic nerves or chiasm. Since these notions have apparently become neurologic dogma, it is interesting to trace their historical development and to examine the evidence on which they are based.

HISTORICAL SURVEY

Their origins lie in the early work of Hitzig¹⁰ (1874), Ferrier¹¹ (1875), Munk¹² (1880) and Schäfer¹³ (1888), who first experimentally

7. Hoppe, H. H.: A Syndrome of the Visuo-Psychic Cortical Area Based on Stable Hallucinations and Defective Visual Association in a Sane Person, *Tr. Am. Neurol. A.*, 1921, p. 247.

8. Jackson, J. H.: Visual Hallucinations Are Sensory Discharges Analogous to Motor Discharges in Epilepsy (Croonian Lecture), *Brain* **11**:179, 1888-1889.

9. Kennedy, F., in discussion on Cushing, H.: Distortion of the Visual Fields in Cases of Brain Tumor, *Tr. Am. Neurol. A.*, 1921, p. 374.

10. Hitzig, G.: Untersuchungen über das Gehirn, Berlin, A. Hirschwald, 1874, vol. 6, p. 276.

11. Ferrier, D.: Experiments on the Brain of Monkeys (Croonian Lecture), *Phil. Tr., London* **165**:433, 1875.

delimited the visual cortex and showed that the projection fibers of the optic pathways were confined to the occipital lobe. From this point of departure it was but another step for the ardent localizationists of this period to interpret all visual phenomena in terms of irritation and destruction of the occipital lobe. Henschen¹⁴ (1890), after an intensive study of this problem, published reports of 16 cases of visual hallucinations which he correlated with irritative lesions of the cortex of the occipital lobe. He was so swayed by his convictions that he disregarded large lesions in other parts of the optic pathways and placed the highest significance on minor lesions in the cortex, which he held entirely responsible for the hallucinations. Westphal¹⁵ (1881), Ingels¹⁶ (1882), Dejerine, Sollier and Auscher¹⁷ (1890) and Wollenberg¹⁸ (1890) had previously published reports of cases of visual hallucinations associated with softenings or tumors in the occipital lobe. In the year preceding Henschen's publication, Jackson and Beevor¹⁹ (1889) reported their now celebrated case. Their patient had attacks ushered in by a foul odor and a sense of suffocation, which was quickly followed by a peculiar dreamy state in which the specter of a little woman dressed in black flitted about the kitchen. Autopsy disclosed a tumor at the tip of the temporal lobe. This case not only defined the uncinat attack but pointed to the fact that visual hallucinations could arise from lesions of the temporal lobe. It remained for Kennedy²⁰ (1911 and 1923) to elaborate this fact and include it in his description of the temporosphenoid syn-

12. Munk, H.: Ueber die Sehphären der Grosshirnrinde, Monatschr. d. k. Preuss. Akad. d. Wissensch., 1880, pp. 485-507.

13. Schäfer, E. A.: An Investigation into the Functions of the Occipital and Temporal Lobes of the Monkey's Brain, Phil. Tr., London (1888) **170**:303, 1889.

14. Henschen, S. E.: Klinische und anatomische Beiträge zur Pathologie des Gehirns, eds. 1-6, Uppsala, Almqvist & Wiksell Boktryckeri, 1890-1922.

15. Westphal, C.: Zur Frage von der Localisation der unilaterale Convulsionen, und Hemianopsie-bedingenden Hirnerkrankungen, Charité-Ann. (1879) **6**:342, 1881.

16. Ingels, B. C.: Par l'apparition des maladies incidentes, Bull. Soc. de méd. de belgique, 1882, no. 2, p. 45.

17. Dejerine, J.; Sollier, P., and Auscher, E.: Deux cas d'hémianopsie homonyme par lésions de l'écorce du lobe occipital, Arch. de physiol. norm. et path. **2**:177, 1890.

18. Wollenberg, R.: Zwei Fälle von Tumor der hinteren Schadelgrube, Arch. f. Psychiat. **21**:778, 1890.

19. Jackson, J. H., and Beevor, C.: Case of Tumor of the Right Temporo-Sphenoidal Lobe Bearing on the Localisation of the Sense of Smell and the Interpretation of a Particular Variety of Epilepsy, Brain **12**:346, 1889-1890.

20. Kennedy, F.: The Symptomatology of Temporosphenoidal Tumors, Arch. Int. Med. **8**:317 (Sept.) 1911; Epilepsy and the Convulsive State, Arch. Neurol. & Psychiat. **9**:567 (May) 1923.

drome. Seguin²¹ (1886) first pointed out that visual hallucinations occurred in the hemianopic field. In view of the cases reported by Reinhard²² (1887), Wilbrand²³ (1890), de Schweinitz²⁴ (1891), Peterson²⁵ (1891) and Burr²⁶ (1906) as well as Seguin in which the visual hallucinations appeared in the hemianopic field, the conviction grew, based on the knowledge that both retinas were represented in each occipital lobe, that hallucinations due to focal lesions of the brain occurred only in the blind field or in the field opposite the lesion.

Cushing²⁷ (1921) presented a study of cases of tumor of the temporal lobe, in 13 of which hallucinations occurred. Cushing believed that they were due to irritation and destruction of the geniculocalcarine bundle in the temporal lobe and that the images were necessarily projected to the contralateral hemianopic field. Although 3 of his patients saw simple unformed images, Kennedy,⁹ in discussing Cushing's paper, remarked that in his opinion crude spectroscopic hallucinations were found only in association with lesions of the occipital lobe and the complex type in association with the lesions of the temporal lobe. This idea gained wide currency and more or less general acceptance. Horrax²⁸ (1923) restudied Cushing's cases and added 4 instances of visual hallucinations associated with tumors of the temporal lobe. Two of these were simple in nature. Horrax and Putnam²⁹ (1932) studied a series of cases of tumor of the occipital lobe and came to the conclusion that only crude visual hallucinations were encountered with such lesions. They discounted 2 cases in which complex hallucinations occurred.

Added to these cases in which the pathologic lesions are verified, there are those cases of epilepsy and migraine accompanied or preceded

21. Seguin, E. C.: A Contribution to the Pathology of Hemianopsia of Central Origin, *J. Nerv. & Ment. Dis.* **13**:1, 1886.

22. Reinhard, C.: Zur Frage der Hirnlocalisation mit besonderer Berücksichtigung der cerebralen Sehstörungen, *Arch. f. Psychiat.* **18**:240, 1887.

23. Wilbrand, H.: Die hemianopischen Gesichtsfeld-Formen und das optische Wahrnehmungszentrum, Wiesbaden, J. F. Bergmann, 1890.

24. de Schweinitz, G. E.: Homonymous Hemianopic Hallucinations with Lesion of the Right Optic Tract, *New York M. J.* **53**:514, 1891.

25. Peterson, F.: Homonymous Hemianopic Hallucinations, *New York M. J.* **52**:241, 1891; **53**:121, 1891.

26. Burr, C.: Visual Hallucinations on the Blind Side in Hemianopia, *Medicine* **12**:491, 1906.

27. Cushing, H.: Distortions of the Visual Fields in Cases of Brain Tumor, *Tr. Am. Neurol. A.*, 1921, p. 374.

28. Horrax, G.: Visual Hallucinations as a Cerebral Localizing Phenomenon, with Especial Reference to Their Occurrence in Tumors of the Temporal Lobe, *Arch. Neurol. & Psychiat.* **10**:532 (Nov.) 1923.

29. Horrax, G., and Putnam, T.: Distortion of Visual Fields in Cases of Brain Tumor—The Field Defects and Hallucinations Produced by Tumors of the Occipital Lobe, *Brain* **55**:499, 1932.

by visual phenomena. The fortification spectrums seen during migrainous attacks with or without an accompanying hemianopia have long been known. That complex elaborate visual experiences have also been observed in association with migrainous attacks is not as well known, although a number of such cases exist in the literature. Weir Mitchell ³⁰ (1887) published 3 cases of migraine in which images of hairy dogs, apparitions of the faces of dead relatives and flowers appeared as an aura or as an accompaniment of the attack. De Schweinitz ³¹ (1889) reported 6 such cases. Ormond ³² (1925) and Engerth, Hoff and Pötzl ³³ (1935) reported similar cases.

Visual auras during an epileptic attack have also been reported by many neurologists of experience. Bennett and Gould ³⁴ (1887) reported an unusual case of a man who sustained a severe injury to the head. A cicatrix resulted which involved the scalp, bone and cortex over the angular gyrus. Pressure on the scar caused sensations of light followed by convulsions.

In addition to these clinical observations, electrical stimulation of the brain has contributed much to the belief that visual hallucinations are only central phenomena. Foerster ³⁵ (1931) by electrical stimulation of areas 17 and 18 caused patients to see flashes of lights and colors before their eyes. Organized images were obtained by stimulation of area 19. However, in spite of the evidence that visual hallucinations may occur in association with lesions of the temporal lobe, no one has succeeded in producing any visual effects by stimulating this lobe.

In view of these many data and in the light of the historical background, it is understandable that the conviction is widely held that visual hallucinations are the result of focal disturbances of the cerebral visuo-sensory and visuopsychic cortex. The overwhelming weight of the many authorities supporting this opinion has submerged the voices objecting to this conception, for it has by no means gone unchallenged. It is curious that so little attention has been paid to these dissenters.

30. Mitchell, S. W.: Neuralgic Headaches with Apparitions of Unusual Character, *Am. J. M. Sc.* **94**:415, 1887.

31. de Schweinitz, G. E.: Headache Associated with Unusual Visual Phenomena, *Univ. M. Mag.* **1**:450, 1889.

32. Ormond, A.: Hallucinations in Sane People, *Brit. M. J.* **2**:376, 1925.

33. Engerth, G.; Hoff, H., and Pötzl, O.: Zur Patho-Physiologie der hemianopischen Halluzinationen, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **152**:399, 1935.

34. Bennett, A. H., and Gould, A.: A Case of Epilepsy of Six Years Duration: Complete Recovery After Surgical Operation on the Skull and Brain, *Brit. M. J.* **1**:12, 1887.

35. Foerster, O.: The Cerebral Cortex in Man, *Lancet* **2**:309, 1931.

Jolly³⁶ (1902) and Pick³⁷ (1904) insisted that visual hallucinations could also be due to irritation of the subcortical visual pathways, and Posey and Spiller³⁸ (1906) stated that "hallucinations of sight may be caused by disturbances of any part of the visual system from the eyeball to the cortical layer of the occipital lobes." Fay³⁹ (1926), Waggoner⁴⁰ (1929) and Terson⁴¹ (1930) have expressed the same opinion. Also, scattered here and there in the literature are cases in which it seems clear that the source of the hallucination lies in the retina. Uthoff⁴² (1899) reported a remarkable case in which visions of colored clouds, birds and angels issued from the stump of an enucleated eye. Another patient with blindness from bilateral detachment of the retina saw dazzling bands of color and "seas of light." Posey and Spiller³⁸ (1906) mentioned a patient with a retinal hemorrhage who suffered visions of horses, pieces of furniture, etc. Ormond³² (1925) recorded another case of retinal hemorrhage, the patient experiencing for some time afterward startlingly clear hallucinations of hideous faces, patterns and lights, though only with the eyes closed. Souter⁴³ (1936) stated that patients in the last stages of glaucoma have strange visions persisting after the eyes are blind.

On the other hand, visual hallucinations occurring with lesions of the optic nerves and chiasm are exceptionally rare! Johnson⁴⁴ (1933), in reviewing the subject of visual hallucinations, stated that "authentic reports of proved cases of visual hallucinations following irritation of the optic tract, chiasm, optic nerve, and retina are practically confined to single cases." Although hallucinations in cases of tabetic atrophy of the

36. Jolly, F.: *Weher Flimmerskatom und Migräne*, Berl. klin. Wchnschr. **39**: 973, 1902.

37. Pick, A.: *The Localizing Diagnostic Significance of So-Called Hemianopic Hallucinations with Remarks on Bitemporal Scintillating Scotomata*, Am. J. M. Sc. **127**:82, 1904.

38. Posey, W. C., and Spiller, W. G.: *The Eye and the Nervous System: Their Diagnostic Relations*, Philadelphia, J. B. Lippincott Company, 1906.

39. Fay, T.: *Visual Hallucinations in Organic Diseases of the Brain*, Arch. Neurol. & Psychiat. **16**:377 (Sept.) 1926.

40. Waggoner, R. W.: *Hemianopsia and Visual Hallucinations*, Arch. Neurol. & Psychiat. **22**:1097 (Nov.) 1929.

41. Terson, A.: *Hallucinations visuelles chez des ophthalmopathes*, Ann. d'ocul. **167**:815, 1930.

42. Uthoff, W.: *Beiträge zu den Gesichtstauschungen (Hallucination, Illusionen) bei Erkrankungen des Sehorgans*, Monatschr. f. Psychiat. u. Neurol. **5**: 240, 1899.

43. Souter: *Neuro-Ophthalmology*, in Berens, C.: *The Eye and Its Diseases*, Philadelphia, W. B. Saunders Company, 1936.

44. Johnson, T. H.: *Visual Hallucinations Accompanying Organic Lesions of the Brain with Special Reference to Their Value as Localizing Phenomena*, Tr. Am. Ophth. Soc. **31**:344, 1933

optic nerve are rather well known, we have been able to find only 6 cases in the literature in which verified tumors of the optic nerves were accompanied by visual phenomena. The first case was reported by Esquirol⁴⁵ in 1845. The patient was a physician who suffered vivid hallucinations of persons and objects for eleven years. While he realized that they were unnatural, some of them seemed so real that at times he reacted to them. He was nevertheless able to keep up his practice. Autopsy revealed a hard calcified tumor the size of a walnut just behind the crista galli and lying on the optic nerves. The nerves were compressed and atrophied (meningioma or craniopharyngioma?). De Schweinitz²⁴ (1891) described the case of a young man in whom several months after a period of morbid sleep and convulsions optic neuritis and a left lateral hemianopia developed. Preceding the onset of the hemianopia the patient had hallucinations in which he saw chairs, tables and other objects in the field which later became blind. He finally became insane and died in an asylum. Autopsy showed a gumma at the base of the brain pressing on the optic tracts in association with gummatus meningitis. Fay and Grant⁴⁶ (1923) reported a case of suprasellar tumor associated with hallucinations. We are again including it in our series. Baruk and Souques⁴⁷ recorded a case of hypophysial tumor in which the left eye was completely blind and there was a temporal hemianopia in the right eye. The patient saw Lilliputian hallucinations in the blind field in his right eye. Beckman and Kubie⁴⁸ (1929) described a patient with a tumor of the hypophysial duct who had hallucinations of lights and colors preceding convulsive attacks, and Uthoff⁴⁹ (1914) stated that one of his patients with an adenoma of the pituitary body had visual hallucinations. No further details are given. So far as experimental stimulation of the optic nerves is concerned, there is little evidence available. It is a common experience of ophthalmic surgeons to have the patients complain of flashes of lights and colors when the optic nerve is cut during enucleation of the eye.^{49a}

These few cases referred to have an importance far beyond their numbers, for they point to the incontrovertible fact that visual hallucina-

45. Esquirol, J. E. D.: *Mental Maladies: A Treatise on Insanity*, translated by E. K. Hunt, Philadelphia, Lea & Blanchard, 1845.

46. Fay, T., and Grant, F. C.: *Lesions of the Optic Chiasm and Tracts with Relation to the Adjacent Vascular Structures*, *Arch. Neurol. & Psychiat.* **9**:739 (June) 1923.

47. Baruk and Souques, cited by Terson.⁴¹

48. Beckman, J., and Kubie, L.: *A Clinical Study of Twenty-One Cases of Tumor of the Hypophyseal Duct*, *Brain* **52**:127, 1929.

49. Uthoff, W.: *Ophthalmic Experiences and Considerations on the Surgery of Cerebral Tumors and Tower Skull*, *Tr. Ophth. Soc. U. Kingdom* **34**:47, 1914.

49a. Quensel, F., in von Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1932, vol. 6.

tions cannot be accounted for entirely on the basis of cortical irritation or of cerebral disease involving the visual centers.

Evidence can be adduced to show that hallucinations of vision occur from lesions at every physiologic level of the neuro-optic apparatus. This concept is apparently at variance with the ideas now current in neurologic circles respecting the clinical significance of visual hallucinations. It is further our intention to point out that hallucinations do not necessarily appear only in the hemianopic fields and further that the complexity of the hallucinations has no localizing value in the diagnosis of focal lesions of the nervous system.

These ideas are not new, but those who have expressed them, in the main, have lacked pathologic verification. To establish this point, 16 cases of tumor implicating only the optic nerves or chiasm have been culled from our collection of hypophysial and parhypophysial tumors. All the patients had visual hallucinations of one kind or another.

From the study of our material it is evident that the presence of hallucinations and their particular character do not depend on the level of the lesion in the neuro-optic system but on psychologic and constitutional factors. The following cases are abbreviated to the essential details.

REPORTS OF CASES

CASE 1.—A 30 year old woman was admitted to the neurosurgical service of the hospital in 1935, complaining of loss of vision and headaches. Her menses had ceased at 19. Four months before admission she began to see "pretty colors" before her eyes. They looked like colored balloons rising from below and floating across her field of vision. Some were green and others red, blue or orange. These were continuous throughout the day and became annoying since they distracted her attention. When she shut her eyes or went into a completely dark room, they were no longer visible. About this time she noted rapidly failing vision.

Neurologic examination showed an alert, intelligent woman. There was bilateral atrophy of the optic nerve. The visual acuity of the right eye was 6/22 and of the left eye 2/60. The visual fields showed a temporal hemianopia in the left eye and a full peripheral field in the right eye with a relative central scotoma. Roentgen examination of the skull showed a large excavated sella turcica.

The operative notes read: "A right transfrontal operation was performed (F. C. G.). The right optic nerve was found humped over a small tumor arising from the pituitary fossa. The left nerve was not seen. The capsule was incised and a good deal of semisolid tumor sucked out. The capsule then collapsed and fell away from the nerves."

The pathologic diagnosis (Dr. B. J. Alpers) was chromophobe adenoma.

After operation the patient no longer complained of the hallucinations, and her vision greatly improved.

Comment.—This case illustrates the occurrence of one of the more simple but not "crude" type of hallucination. The obvious lesion was a small adenoma of the pituitary body compressing the optic nerves. The patient was perfectly alert and aware that her visual experiences were

hallucinatory. The hallucinations were not localized in the blind portions of the field but occurred directly in line with central vision.

CASE 2.—A 31 year old woman was admitted to the neurosurgical service of the hospital in 1931, with the history of gradually failing vision for eight years and rapid failure in the five months prior to admission. For a number of years since her vision began to fail she had noticed unusual visual experiences. Many times daily she had attacks in which showers of brightly colored sparks danced in front of her eyes. At night or in the dark they lost their color and appeared as flashes of white lights.

Neurologic examination gave negative results except for the visual findings. The patient was intelligent and cooperative. There was bilateral atrophy of the optic nerve. Visual acuity was 1/60 in the right eye and 6/22 in the left eye. The visual fields showed a bitemporal hemianopia with macular encroachment on the right. Roentgen examination of the head showed a large and eroded sella turcica.

A transfrontal operation was performed (F. C. G.). According to the operative notes "a large bluish cyst was found lying beneath the right optic nerve. It was aspirated, whereupon it collapsed. The capsule was almost completely removed." The patient died of complications on the third postoperative day.

The pathologic diagnosis was chromophobe adenoma (Dr. B. J. Alpers).

Comment.—In this case the tumor was small and was entirely underneath the nerves. The hallucinations were not lateralized or restricted to the blind portions of the field. The patient was clear and had perfect insight into the hallucinatory character of her visual experiences.

CASE 3.—A 30 year old man was admitted to the neurosurgical service of the hospital in 1928, with the history of severe headaches for three years associated with progressive enlargement of his features and of his hands and feet. There had been no visual complaints. Drowsiness had been marked, and he had lost his libido.

Examination showed a young man with an advanced acromegaly. The visual fields were full, the visual acuity was normal and the disks were normal. The pituitary fossa was extremely large and eroded.

Because of the severe headaches, a right transfrontal craniotomy was performed by Dr. Charles H. Frazier. His notes read: "Anterior to the chiasm but not quite reaching it was a plum colored tumor. The optic nerves did not appear to be pressed on. The capsule was incised and a considerable amount of tumor sucked out." The patient made an uneventful recovery and was discharged free of complaints.

Three months later he returned complaining of transitory episodes of blurring of vision and peculiar visual experiences. Several times daily he would suddenly see streaks of colors flashing before his eyes. Sometimes there were bursts of colors like skyrockets going off in all directions. They were seen directly in front of him. Visual fields taken at this time showed a temporal hemianopia to red in the right eye and a relative paracentral scotoma. Visual acuity was 6/6 in the right eye and 6/9 in the left eye. He was given roentgen treatments, with general improvement, but unfortunately no notes were made concerning the hallucinations.

The pathologic diagnosis was eosinophilic adenoma (Dr. B. J. Alpers).

Comment.—This case is especially interesting because the appearance of visual hallucinations coincided with the appearance of evidence of injury to the right optic nerve. This was undoubtedly due to the regrowth of the tumor, possibly stimulated by the previous operation. Before the operation there had not been any hallucinations or visual impairment, and the first operation demonstrated that the tumor was not pressing on the optic nerves. Although there was only evidence that the right nerve was injured, the color visions were seen in the fields of both eyes.

CASE 4.—A man 54 years of age was admitted to the neurosurgical service of the hospital in 1935. For twenty years he had had pains around his right eye, which increased slowly in severity as time went on. There had been gradual failure of vision for seventeen years. The left eye became blind in 1924, and the right eye failed subsequently. For many years he had noted from time to time flashes of colors in front of his eyes. They looked like multicolored lights. On many occasions the field in front of him would become bright red or bright green, or else alternate waves of color would succeed one another. At other times he would see complicated colored geometric patterns, which would weave and change so that it seemed to him as if he were looking into a child's kaleidoscope. The geometric patterns might be replaced by numerals or by colored spots.

Examination showed an intelligent, clear and cooperative middle-aged man. The left eye was totally blind, and vision was restricted to perception of light in the right eye. There was marked bilateral atrophy of the optic disk. Roentgen examination showed marked enlargement and erosion of the sella turcica.

A left transfrontal craniotomy was performed by Dr. Charles H. Frazier. His notes read: "The tumor was found partly within and partly without the sella. The main mass of the tumor was pushing upward on the chiasm. The capsule was incised, as much of the tumor removed as possible, and the nerves freed by dissection." The patient recovered and was discharged, but his vision did not improve. There was no further note concerning his visual hallucinations.

The pathologic diagnosis was chromophobe adenoma (Dr. B. J. Alpers).

Comment.—These hallucinations were more elaborate than those in the preceding cases, for design and pattern are added. The lesion was confined to the optic nerves and chiasm. The patient was mentally clear and aware of the fact that his experiences were hallucinations. Though vision was lost completely in one eye and only an undefinable fragment of a field remained in the other, the hallucinations were seen throughout the entire "field" of vision.

CASE 5.—A 37 year old man was admitted to the neurosurgical service of the hospital in 1937. There had been headaches for seven months and failing vision for four years. For the two years before admission he had noted odd visual experiences. From time to time, especially if he looked into the distance, he would see "wheels" spinning before his eyes. They were not very annoying and passed away in a few minutes.

The patient was intelligent and alert and had worked as a bank cashier until his admission to the hospital. There was a mild degree of bilateral atrophy of

the optic disk. Visual acuity was 6/12 in the right eye and 6/9 in the left eye. The fields, however, showed a bitemporal hemianopia sparing the maculas. The sella turcica was enlarged.

A right transfrontal craniotomy was performed (F. C. G.). The operative notes read: "A dark plum colored tumor was found spreading out beneath the chiasm and pushing it up. The right frontal lobe was resected and the largest part of the tumor removed."

The pathologic diagnosis was chromophobe adenoma (Dr. B. J. Alpers).

The patient had a stormy postoperative course but recovered, with greatly improved fields. A year later he was back at work in the bank and had no visual complaints. He did not experience any more hallucinations after his operation.

Comment.—The notes on this patient are regrettably brief, but it seems as if the hallucinations he experienced were fairly complicated and displayed a recognizable form as well as motion. As far as he could determine, they were seen directly in front of him.

CASE 6.—A 21 year old man was admitted to the neurosurgical service of the hospital in 1913, with the complaint of violent headaches for two years and failure of vision for eighteen months. Nine weeks before admission he began to lose sight rapidly. For many months before this, however, he had noticed waves of vivid colors sweeping over his fields of vision. The colors were either intensely red or intensely green. Frequently he would see a glowing white light toward the right side of his field. These attacks were episodic and passed away after a few minutes but occurred many times daily. They continued after he had lost all vision and were present on his admission to the hospital.

Neurologic examination showed a clear and cooperative patient who was totally blind in both eyes. No perception of light was present. The sella turcica was large and eroded and the sphenoid bone encroached on. There was advanced bilateral atrophy of the optic nerve.

A right transfrontal operation was performed by Dr. Charles H. Frazier in the hope of restoring some vision. His notes read: "The sella turcica was found full of a soft, reddish tumor which surrounded the optic nerves and chiasm. A large amount was removed, and the nerves were widely freed." The patient was sent home with relief of headaches but with no return of vision. No further hallucinations were noted.

The pathologic diagnosis was malignant adenoma of the pituitary body.

Comment.—The interesting fact about this patient was the presence of visual hallucinations not only while he was losing vision but even after he had become completely blind. Even though vision was lost, enough of the optic fibers central to the tumor must still have remained irritable and capable of conducting excitations, otherwise the visual sensation could not have appeared in consciousness. That the tumor was responsible for the excitations is suggested by the fact that after release of the optic nerves they ceased. The appearance of the light at the right side of the field indicates that visual hallucinations may be lateralized in the presence of complete blindness.

CASE 7.—A 23 year old woman was admitted to the neurosurgical service of the hospital in 1927. There had been cessation of menses for eight years. Five

years before admission her breasts enlarged and began to secrete milk copiously. For four years they remained large and tender. Lactation was free. Headaches had been present for seven years, polydipsia and polyuria for three years and failing vision for one year. Two months before admission, while lying down one afternoon, she was suddenly amazed to see the room populated. She was aware that the persons she saw could not be real. She did not recognize any of them, although they were vivid and walked about her bed. "I could see them distinctly, but I didn't know who they were." The spectral persons filled her room for quite a while and then faded away. This happened several other times and was usually preceded by a dizzy attack.

Examination showed an alert young woman who gave her own history intelligently. There were marked features of dysendocrinism. Visual acuity was 6/12 in the right eye and 6/9 in the left eye. There was papilledema of 4 D. in the right eye and 1 D. in the left eye, but the right visual field showed a temporal cut down to 30 isopters. The sella turcica was large and eroded.

A right transfrontal craniotomy was performed by Dr. Charles H. Frazier. His notes read: "The lesion presented as a large bluish cyst under the chiasm, extending between the arms of the optic nerves. The left optic nerve was flattened against the wall of the cyst. An incision into the capsule was made and the contents aspirated and curetted out. The capsule was freed from both nerves and as much as possible removed."

The pathologic diagnosis was chromophobe adenoma.

The patient left the hospital much improved. No further note appears in the follow-up records concerning her hallucinations.

Comment.—This case demonstrates a highly elaborate and complex type of hallucination occurring in an alert, critical and mentally clear patient. The lesion of the optic pathways was confined to the optic nerves and chiasm. Although there was only a field cut in the temporal side of the right eye, the apparitions were seen over the entire field of vision.

CASE 8.—A boy 10 years old was admitted to the neurosurgical service of the hospital in 1935. There had been cessation of body growth since the age of 8, gradual failure of vision and headaches.

Examination showed an obvious hypophysial dwarf. Only perception of light was retained in the left eye, and there was a white atrophic disk. Vision in the right eye was 6/6, the field was full and the disk was normal. The sella turcica was eroded completely away, and there were suprasellar calcifications. The boy was bright and alert and had been continuing in school.

A right transfrontal operation was performed (F. C. G.). The operative notes read: "A purplish tumor was found lying behind and stretching the left optic nerve. Although the tumor was suprasellar, it was under the chiasm and nerve. Fifteen cubic centimeters of 'motor oil' fluid was removed and as much as possible of the collapsed cyst resected." The postoperative course was uneventful except for a slight rise in temperature and a convulsion on the fourteenth postoperative day. The patient was up in a chair and about to go home, when one evening he began to have hallucinations. He saw a boy dressed in green with a bow and a quiver of arrows, and many other figures and persons. The house officer's notes read: "The patient was quite rational about these hallucinations and realized their fictitiousness, but at times they appeared so real that he could not help but be

fearful and react to them." The hallucinations kept up for three days and so interfered with his sleep that sedatives had to be administered. They gradually faded away, and the boy was discharged.

The pathologic diagnosis was tumor of the hypophysial duct.

Comment.—It is somewhat difficult to understand why these hallucinations should have appeared so long after operation or why they did not appear preoperatively. Possibly the explanation is that reparative processes about the nerves, absorption of blood or organization of connective tissue stimulated the optic nerves. The significant fact is that the hallucinations appeared against a mentally clear background, and the only lesion of the neural portion of the optic pathways was a tumor pressing on the chiasm. They were apparently seen over the entire visual field even though the patient was blind in one eye.

CASE 9.—A 17 year old girl was admitted to the neurosurgical service of the hospital in 1927, with a history of headache for seven years and visual failure for six years. Shortly after she noticed failing vision she had an episode of visual hallucinations that lasted three weeks. These seemed to occur only in the dark. When she was in bed she would see pictures of various persons appearing and disappearing on the walls of her room. She recognized the phantom faces as belonging to popular movie stars. During all these attacks, which occurred every night, she was perfectly lucid and would describe the hallucinations to members of her family at great length. There were a number of such episodes. She also complained that while in a dark room she would see a bright white light glowing in front of her. Coincident with the onset of her hallucinations, she noted a "scum" over the lateral areas of her fields of vision.

Neurologic examination showed a bright, well oriented and alert young woman. There was bilateral atrophy of the optic nerve. The visual acuity was 6/15 in the right eye and 6/9 in the left eye. The visual fields showed bitemporal hemianopia. The sella turcica was greatly enlarged and eroded.

A right transfrontal craniotomy was performed by Dr. Charles H. Frazier. His operative notes read: "A cystic tumor was found beneath the right optic nerve. This was aspirated and the wall of the cyst removed." Vision in the right eye rapidly improved. Four months later a left frontal craniotomy was performed, and the portion of the tumor which projected beneath the left optic nerve was removed. An uneventful recovery was made.

Comment.—The patient apparently had so-called simple hallucinations of light as well as the more elaborate variety, consisting of recognizable faces on the wall. It is difficult here as well as in the other cases to explain why the hallucinations should be episodic when the lesion is constantly present. However, this differs not at all from the episodic nature of hallucinations noted in cases of hemispheric tumor. The factors on which this is dependent are unknown, but that they do occur in this fashion is a matter of common clinical knowledge. No lateralization of the images was present in this case despite the fact that the temporal fields were obliterated.

CASE 10.—A 20 year old youth was admitted to the neurosurgical service of the hospital in 1921, with a history of headaches for six months, associated with rapid failure of vision.

The neurologic findings were entirely negative except for the eyes. The patient was clear and cooperative and gave his own history. There was bilateral papilledema of 4 D. and a homonymous left hemianopia. Roentgen examination of the skull showed a "top normal" sella turcica with slight erosion of the posterior clinoid process. A ventriculogram showed a "suspicious defect" in the posterior horn of the right ventricle. On the basis of this and the hemianopia, a large craniotomy was performed on the right side by Dr. Charles H. Frazier. Nothing was found, and the wound was closed after provision was made for a decompression. The patient recovered from this procedure and was out of bed and walking around two weeks later. He then began having hallucinations. Dr. Walter Freeman, then house officer, supplied excellent notes on this case: "The hallucinations occurred with the eyes open, but more particularly when they were shut. They appeared only to exist toward the right side (blind field). They were persistent but not constant, and they were abolished by attempts at fixation. The hallucinations were accompanied by no other emotion than annoyance, and they were clearly realized to be imaginary. The type of hallucination varied; the patient saw trees, pictures on the walls, colored but portraying no definite scene that he could recall as having seen before. He saw a complicated pattern on the floor, like a carpet; also boxes and crates on the floor. He saw a nonexistent building with a scaffolding around it. Sometimes the patients on the other side of the ward appeared to have hair 3 feet long. The hallucinations lasted a week, during which the patient was depressed because of the annoyance of the images and because of the anxiety they caused him."

A small infection on the edge of the wound persisted and prevented exploration of the sellar region. One month after the operation the infection extended, and the patient died of meningitis.

Autopsy revealed a small solid tumor, about the size of a walnut, resting on the posterior edge of the optic chiasm. The chiasm was flattened. In addition, it had been pushed forward so that the internal carotid arteries on either side had deeply grooved the optic nerves. The anterior communicating artery had notched the upper surface of the chiasm. This case was reported by Fay and Grant⁴⁰ in 1923, and the hemianopia was accounted for by the arterial compression of the optic nerves.

The pathologic diagnosis was adamantinoma.

Comment.—This case shows how a combination of homonymous hemianopia and visual hallucinations due to a chiasmal tumor can simulate a tumor of the temporal or the occipital lobe if the conception is held that visual hallucinations are purely central phenomena. The decompression possibly allowed certain shifting of structures to take place in the region of the tumor sufficient to stimulate the optic fibers and provoke hallucinations. Again the lucidity of the patient and his insight into the hallucinatory quality of his visual experiences exclude any serious disturbance of the brain, which, on its own account, may have given rise to visual misinterpretations.

CASE 11.—A man 39 years old was admitted to the neurosurgical service of the hospital in 1929. There had been progressive enlargement of his facial

features and of his extremities for fourteen years. Vision had failed for three years, and his left eye had been blind for eighteen months. Headaches and loss of libido had existed for one year.

Examination showed advanced acromegaly. The left eye retained perception of light only, and the optic disk was atrophic. Vision in the right eye was 6/15, and there was a temporal hemianopia. The pituitary fossa was enormously enlarged.

A right transfrontal operation was performed by Dr. Charles H. Frazier. His notes read: "The bluish wall of a cyst presented prechiasmally between the arms of the optic nerves. When incised, it collapsed and appeared empty. Part of the capsule was removed." The patient made an uneventful recovery.

The pathologic diagnosis by Dr. B. J. Alpers was eosinophilic adenoma.

The right eye began to improve after the operation, but the left eye lost perception of light at the end of a year. Coincident with this, the patient began to experience visual hallucinations of various kinds. Every week or two a bright white light would suddenly appear in front of his blind left eye. In the course of a few seconds it would expand until it was large and the glare was intense. It then began to fade and disappear. It would be followed by a red light that would go through the same process of expanding, growing bright and then fading. In succession, a green, blue or orange light would appear. Such an attack lasted about five minutes. The lights were never noted in front of his right, or good, eye. At the time of writing he is able to go about his work undisturbed except for the annoyance of the experience. More distressing are his night "visions." Almost every night when he retires "visions" appear. They occur more often when he closes his eyes, but not necessarily. The field in front of "both" his eyes becomes bright and luminous, and in this luminous field patterns appear. They are complicated designs in color which remind him of wallpaper. They whirl, shift and change, and different patterns succeed one another. They last for hours and prevent his sleeping. They have persisted ten years since his operation and have worried him greatly because they make him doubt his sanity. They so interfere with his rest that his chief request is for "something to make him sleep." He is aware of the fact that they are "not real" and that they "have no business to be there." He is alert and intelligent and is working steadily. His left eye is totally blind, and the disk is atrophic. His right eye has vision of 6/6 — 4. There is a superior temporal quadrantanopia.

Comment.—In this case there are two different kinds of visual experiences, one which is simple in nature and entirely unilateral and the other which is elaborate and seen (sic) over the entire field. It is evident that pressure on the optic nerves is still present. This is indicated by the gradual loss of perception of light in the left eye and by the fact that the sella turcica has enlarged from 24 by 18 mm. to 26 by 19 mm. in the past ten years. The patient has a perfectly intact intelligence and personality, in spite of which he sees these various hallucinations. The unilateral hallucinations in the left eye are doubtless due to pressure irritation of the left optic nerve, which in spite of its apparent complete atrophy must contain sufficient viable central fibers to conduct excitations to the brain. In this instance the phenomenon is analogous to the so-called "phantom limb." The presence of hallucinations, which

seem to be in both fields, indicates pressure on the chiasm, with psychic projection of the percepts elaborated from the excitations into the fields of both eyes.

CASE 12.—A 23 year old man was admitted to the neurosurgical service of the hospital in 1936. There had been progressive enlargement of his facial features and extremities for five years and severe headaches for three years. About a year before admission he began to suffer from unusual visual experiences. A glowing purple "bubble" would appear in front of his eyes, small at first, but enlarging rapidly until it seemed to "burst." This would be succeeded by another that went through the same sequence. This occurred a number of times before the attack was over. The episodes took place three or four times weekly. At other times he would see what appeared to be a whirling wheel before his eyes, which would enlarge, seem to come close, then grow smaller and recede until it vanished. Occasionally he would see groups of iridescent bubbles ascending before his eyes. They seemed to start at the lower portion of his field and float upward. At the upper portion of his field they burst "just like soap bubbles." All these visual impressions could be abolished by closing and rubbing the eyes for a few minutes.

Examination showed a young man with well marked acromegaly. The sella turcica was enlarged in all diameters. Although he had not complained of visual loss, the visual acuity was 6/30 in the right eye and 6/22 in the left eye. The visual fields showed bitemporal contraction to the fortieth isopter and bilateral paracentral scotomas. There was a bitemporal hemianopia to red. The patient had, on examination, a perfectly clear mentality.

A right transfrontal craniotomy was performed (F. C. G.). The notes read: "A fairly small reddish tumor lay between the optic nerves and seemed to make only slight pressure on them. The capsule was incised, a portion resected and the interior curetted out."

The patient made an uneventful recovery, and his fields widened out to normal. Visual acuity improved to 6/6 in the right eye and 6/15 in the left eye. At the time of writing it is two years since his operation, during which time he has received intensive roentgen treatment. His visual hallucinations have persisted. Four to six times daily he sees wavy black lines darting across his field of vision. Sometimes they appear exactly like the bursting of a skyrocket with colored light shooting in all directions from a central point and drifting out of sight. He still sees the image of a spinning wheel in front of him at times but no longer sees the glowing purple lights. As far as he can tell the images appear to be directly in front of him. His present visual acuity is 6/6 in the right eye and 6/21 in the left eye. His fields remain full to form though showing a bitemporal hemianopia to color.

The pathologic diagnosis was eosinophilic adenoma (Dr. B. J. Alpers).

Comment.—The hallucinations of this patient varied from the most simple to elaborate types. There was no correspondence between the blind areas of vision and the portion of the fields in which he saw the hallucinations. Although the fields widened after the operation, there is no doubt that some type of irritation still remains in or about the optic nerves. The only lesion of the optic pathways was the tumor. The visions were observed by an alert and critically intelligent person who clearly realized that the images were unreal.

CASE 13.—A 56 year old woman was admitted to the neurosurgical service of the hospital in 1935. There had been failure of vision in her left eye for one year and in her right eye for four months. From the onset of her visual difficulties she began to experience peculiar visual attacks. On many occasions she would suddenly see her fields of vision before her studded with blue stars. These would last a few minutes and fade out. On other occasions everything in front of her would "go gray," and on this gray field were glowing lavender stars. This vision would last a little while and fade away. Occasionally little gray squares would appear in front of her eyes. These hallucinations persisted until her admission and occurred at least once daily. There had been sharp, shooting pains in her head for several months.

Examination revealed an alert, intelligent middle-aged woman. Only perception of light was present in the right eye, and visual acuity was 1/60 in the left. Examination of the visual fields showed that only small undelimitable islands of nasal vision remained. There was advanced bilateral atrophy of the optic disk. The sella turcica was "dished out," and there were fine suprasellar calcifications.

A right transfrontal craniotomy was performed (F. C. G.). The operative notes read: "The right optic nerve was found knuckled over a tumor. Aspiration yielded motor oil fluid. As much of the capsule and tumor was removed as was possible." The patient made an uneventful recovery, the vision in her right eye returning to 6/9. There was no further mention of visual hallucinations in her follow-up notes.

The pathologic diagnosis was tumor of the hypophysial duct.

Comment.—Although the patient had only small islands of vision in her nasal fields and although both maculas were encroached on, the images were seen all over her "fields" of vision. The tumor pressed upward on the right optic nerve and the chiasm, although it arose from without the sella turcica. The patient was clear mentally and appreciative of the fact that these phenomena were hallucinatory.

CASE 14.—A man 34 years old was admitted to the neurosurgical service of the hospital in 1936. There had been failure of vision in his left eye for two years, noticed first in the lateral field but progressing to blindness in one year. Then vision began to fail in the right eye. About this time the patient began to see a "glowing white star" toward his right side. The star remained there continually whether he was in the light or in a completely dark room. It remained and persisted even when vision was lost in that portion of his field. It had glowed there constantly for a year when he entered the hospital. There had been a marked loss of weight associated with a loss of body hair and of libido.

Examination showed a mentally clear young man who looked much younger than his years. There was a distinctly feminine habitus with almost complete loss of body hair. His voice was a high falsetto. The left eye was totally blind, and vision was reduced to perception of movement in the right eye. The sella turcica measured 35 by 25 mm., and there were supersellar calcifications.

A right transfrontal craniotomy was performed (F. C. G.). The operative notes read: "The right optic nerve and the right carotid artery were displaced by a purplish cyst. It was aspirated, a portion of its capsule was removed, and the nerves were dissected free." The patient made an uneventful recovery but had no return of vision.

The pathologic diagnosis was tumor of the hypophysial duct.

Comment.—The curious feature of this case was the constancy of the glowing light toward the right side of the patient, becoming visible as vision was lost in the right temporal field. In our other cases the visual phenomena have been more or less paroxysmal. The patient was aware that this glowing star represented some unusual kind of visual experience, since it was part of his complaint.

CASE 15.—An 18 year old girl was first admitted to the neurosurgical service of the hospital in 1923. There had been complaints of diplopia, headache, amenorrhea, and bitemporal loss of vision. A cyst of the pituitary body was diagnosed and partly removed at operation. Over the course of the following thirteen years the patient had frequent roentgen treatments and two more transfrontal operations with evacuation of the cyst and partial removal of the capsule for recurrent visual failure. In 1936 she was again readmitted to the hospital for visual failure. On this examination she was blind in her left eye and had vision of only 6/22 in her right eye associated with a temporal hemianopia. A fourth operation was performed. A few small adhesions were found around the remnants of the left optic nerve. Behind it and under the chiasm was a yellowish white tumor. The capsule was partly removed again and the contents sucked out.

After the operation the patient was completely blind! This could not be accounted for except on the assumption that the vascular supply to the nerves was somehow damaged. The patient felt well otherwise and was discharged. From this time on the patient began to experience visual phenomena. Even while in the hospital she thought at times that she was regaining her vision since she saw "light," and her fields seemed to be illuminated. Neither of her pupils gave the least reaction to light, nor was she able to perceive the strongest lamp held in front of her eyes. She learned Braille and typing, and in writing to the nurses who cared for her while she was in the hospital she would state that she was sure her sight was returning since frequently she saw light, and on one occasion she wrote that she saw moving objects, like snakes. She also reported that she was seeing colors. She soon began to complain to her parents that she was seeing visions all the time composed of persons, scenes and objects. They were more or less constantly before her view and annoyed her as well as prevented her from sleeping. They interfered with her activities of reading Braille and writing. She was apparently mentally alert at this time since she was carrying on a large correspondence. She reconciled herself to the fact that these visions did not indicate a return of sight and knew that they were visionary and unreal. Her parents were so distressed at her suffering with these visions that they wrote for help, which, unfortunately, could not be given. As time went on her attitude toward her visions changed, and she began to act as though they were real. This was more than a year after the hallucinations appeared. She started to hold conversations with them and lost her appreciation of their unreality. At the time of writing, three years after operation, she is still alive. Her parents write that she is constantly seeing visions and talking a great deal of the time to the faces and figures of her friends and acquaintances that compose them. At times she is fearful of what she sees, and at other times she obviously enjoys them. Her images are highly detailed, and in her more lucid and accessible moments she describes them to her parents. She is confused a large part of the time. Her father questioned her at our request and wrote us that the visions are seen mostly toward the left side.

CASE 13.—A 56 year old woman was admitted to the neurosurgical service of the hospital in 1935. There had been failure of vision in her left eye for one year and in her right eye for four months. From the onset of her visual difficulties she began to experience peculiar visual attacks. On many occasions she would suddenly see her fields of vision before her studded with blue stars. These would last a few minutes and fade out. On other occasions everything in front of her would "go gray," and on this gray field were glowing lavender stars. This vision would last a little while and fade away. Occasionally little gray squares would appear in front of her eyes. These hallucinations persisted until her admission and occurred at least once daily. There had been sharp, shooting pains in her head for several months.

Examination revealed an alert, intelligent middle-aged woman. Only perception of light was present in the right eye, and visual acuity was 1/60 in the left. Examination of the visual fields showed that only small undelimitable islands of nasal vision remained. There was advanced bilateral atrophy of the optic disk. The sella turcica was "dished out," and there were fine suprasellar calcifications.

A right transfrontal craniotomy was performed (F. C. G.). The operative notes read: "The right optic nerve was found knuckled over a tumor. Aspiration yielded motor oil fluid. As much of the capsule and tumor was removed as was possible." The patient made an uneventful recovery, the vision in her right eye returning to 6/9. There was no further mention of visual hallucinations in her follow-up notes.

The pathologic diagnosis was tumor of the hypophysial duct.

Comment.—Although the patient had only small islands of vision in her nasal fields and although both maculas were encroached on, the images were seen all over her "fields" of vision. The tumor pressed upward on the right optic nerve and the chiasm, although it arose from without the sella turcica. The patient was clear mentally and appreciative of the fact that these phenomena were hallucinatory.

CASE 14.—A man 34 years old was admitted to the neurosurgical service of the hospital in 1936. There had been failure of vision in his left eye for two years, noticed first in the lateral field but progressing to blindness in one year. Then vision began to fail in the right eye. About this time the patient began to see a "glowing white star" toward his right side. The star remained there continually whether he was in the light or in a completely dark room. It remained and persisted even when vision was lost in that portion of his field. It had glowed there constantly for a year when he entered the hospital. There had been a marked loss of weight associated with a loss of body hair and of libido.

Examination showed a mentally clear young man who looked much younger than his years. There was a distinctly feminine habitus with almost complete loss of body hair. His voice was a high falsetto. The left eye was totally blind, and vision was reduced to perception of movement in the right eye. The sella turcica measured 35 by 25 mm., and there were supersellar calcifications.

A right transfrontal craniotomy was performed (F. C. G.). The operative notes read: "The right optic nerve and the right carotid artery were displaced by a purplish cyst. It was aspirated, a portion of its capsule was removed, and the nerves were dissected free." The patient made an uneventful recovery but had no return of vision.

The pathologic diagnosis was tumor of the hypophysial duct.

Comment.—The curious feature of this case was the constancy of the glowing light toward the right side of the patient, becoming visible as vision was lost in the right temporal field. In our other cases the visual phenomena have been more or less paroxysmal. The patient was aware that this glowing star represented some unusual kind of visual experience, since it was part of his complaint.

CASE 15.—An 18 year old girl was first admitted to the neurosurgical service of the hospital in 1923. There had been complaints of diplopia, headache, amenorrhea, and bitemporal loss of vision. A cyst of the pituitary body was diagnosed and partly removed at operation. Over the course of the following thirteen years the patient had frequent roentgen treatments and two more transfrontal operations with evacuation of the cyst and partial removal of the capsule for recurrent visual failure. In 1936 she was again readmitted to the hospital for visual failure. On this examination she was blind in her left eye and had vision of only 6/22 in her right eye associated with a temporal hemianopia. A fourth operation was performed. A few small adhesions were found around the remnants of the left optic nerve. Behind it and under the chiasm was a yellowish white tumor. The capsule was partly removed again and the contents sucked out.

After the operation the patient was completely blind! This could not be accounted for except on the assumption that the vascular supply to the nerves was somehow damaged. The patient felt well otherwise and was discharged. From this time on the patient began to experience visual phenomena. Even while in the hospital she thought at times that she was regaining her vision since she saw "light," and her fields seemed to be illuminated. Neither of her pupils gave the least reaction to light, nor was she able to perceive the strongest lamp held in front of her eyes. She learned Braille and typing, and in writing to the nurses who cared for her while she was in the hospital she would state that she was sure her sight was returning since frequently she saw light, and on one occasion she wrote that she saw moving objects, like snakes. She also reported that she was seeing colors. She soon began to complain to her parents that she was seeing visions all the time composed of persons, scenes and objects. They were more or less constantly before her view and annoyed her as well as prevented her from sleeping. They interfered with her activities of reading Braille and writing. She was apparently mentally alert at this time since she was carrying on a large correspondence. She reconciled herself to the fact that these visions did not indicate a return of sight and knew that they were visionary and unreal. Her parents were so distressed at her suffering with these visions that they wrote for help, which, unfortunately, could not be given. As time went on her attitude toward her visions changed, and she began to act as though they were real. This was more than a year after the hallucinations appeared. She started to hold conversations with them and lost her appreciation of their unreality. At the time of writing, three years after operation, she is still alive. Her parents write that she is constantly seeing visions and talking a great deal of the time to the faces and figures of her friends and acquaintances that compose them. At times she is fearful of what she sees, and at other times she obviously enjoys them. Her images are highly detailed, and in her more lucid and accessible moments she describes them to her parents. She is confused a large part of the time. Her father questioned her at our request and wrote us that the visions are seen mostly toward the left side.

Comment.—This exceptional case raises many problems of interpretation. Two things stand out; first, that her visual hallucinations for a long while were associated with an alert and critical intellect and, secondly, that they appeared after her optic nerves had been injured severely enough to cause blindness. Certainly some stimulus must exist to initiate any sensation or perception. In this instance it is presumed to be irritation of the proximal ends of the optic nerves by fibrotic reaction or compression by fibrosis. In spite of the total blindness, enough fibers must remain functionally intact, because otherwise the excitations could not be presented to consciousness. This is also indicated by the fact that the visual hallucinations of lights and colors began while she was still in the hospital and before complete degeneration of the nerves would be expected to occur. An analogous phenomenon is noted in a completely anesthetic area in which dysesthesias may be experienced. The late stage in which all sense of reality was lost and the patient no longer appreciated the nature of the images can be ascribed only to general disturbance of the brain, likely due to growth of the tumor into the frontal lobes. The source of the visions, however, no matter how interpreted by the patient and with what little insight, is still likely due to the irritation of the optic nerves. This cannot be said with certainty since increased intracranial pressure may have set in, and the visual tracts may be irritated at other points or the entire function of the brain disturbed by it.

CASE 16.—A man 59 years old was admitted to the neurosurgical service of the hospital in 1937, complaining of loss of vision. Five and one-half months before admission he noted blurring and indistinctness of vision. His sight became rapidly diminished, especially in the left eye. There had been loss of libido within the year before admission. No headaches were complained of.

On examination the patient was alert, intelligent and asthenic. Visual acuity was 6/6 in the right eye and 6/30 in the left eye. There was a moderate degree of bilateral atrophy of the optic nerve. Perimetric fields showed only a small upper nasal island of vision remaining in the left eye and a temporal hemianopia in the right eye. Roentgen examination of the skull disclosed a large and eroded sella turcica.

A left transfrontal craniotomy was performed. The operative notes read (F. C. G.): "The left optic nerve was exposed and found to be pushed forward by a bluish gray tumor. This was incised, and a large amount gutted out from the interior." The patient did well postoperatively, but it was noted on the second day that he was blind. This raised the suspicion of a postoperative hemorrhage, and the flap was reelevated. The operative notes on this occasion read: "The tumor bed was found filled with clot, which had distended the tumor. This was removed together with a large additional amount of tumor tissue, so that the right optic nerve was exposed. Both optic nerves seemed to be undamaged." The patient had a stormy postoperative course, from which he finally recovered. Vision, however, did not return, and he has remained permanently blind.

The pathologic diagnosis was chromophobe adenoma (Dr. B. J. Alpers).

After discharge from the hospital, the patient made an adjustment to his blindness and took up Braille. When seen in the follow-up clinic a short time later he began to ask questions concerning the visions he was having. A short time after leaving the hospital he began to see various images, which at first led him to suspect that vision was returning. He would see stars shining in the sky and clouds moving before his eyes, and instead of experiencing the expected darkness his fields of vision were brightly illuminated. It seemed to him that most of the time he was looking at a white surface studded with rounded knobs, like cobblestones. Eventually he reconciled himself to the fact that these visions were phantasies and that his sight was not returning. After a short while, however, his visual experiences became richer, more varied and detailed. He began to see landscapes, country and mountain scenes, none of which, however, were familiar. Houses and buildings were seen. When questioned about the amount of detail, he stated that he could make out the walls and roofs, doors and windows but not much else. He saw at times animals, men riding horseback and children playing and on one occasion a vivid picture of a mother nursing her baby. These visions were seen most of the day and passed before him in panoramic procession. He was perfectly aware that they were hallucinatory and was somewhat embarrassed to describe them too fully lest the examiners think him "crazy." The patient regaled the examiner with a running account of the images he was seeing as he sat in the clinic. He described scenes, faces, smoke drifting across a landscape, the moon shining in a star studded sky and a number of other objects. The images were seen directly in front of him, though they moved across his view from left to right.

Examination disclosed that all vision, including perception of light, was absent. The left pupil reacted slightly to direct light; the right did not react. There was bilateral advanced atrophy of the optic nerve. Mental examination showed the patient to be mentally normal if not of superior intellect. Judgment and insight were good, and his comment on his visions were witty and amusing. His family vouched for his mental integrity and spoke at length of the wide range of his interests.

Comment.—In this case, as in the preceding one, visual hallucinations began after total blindness occurred. In both these cases there is ample evidence of injury to the optic nerves. The operative findings indicate that the lesion was confined to the optic nerves. There is no question in this case of injury to other parts of the brain. Neurologically and mentally, the patient is entirely normal, with the exception of blindness. He is perfectly aware of the hallucinatory character of the images he sees. Though perception of light is absent, the activity of the left pupil to light establishes the fact that there are some fibers in the optic nerve which are still physiologically active. This is, on a priori grounds, necessary, for else excitations arising in the optic nerves could not be transmitted so that they would appear in consciousness. The appearance of these hallucinations postoperatively is probably due to stimuli set up by widespread reparative or destructive processes occurring in and about the optic nerves. The images are seen as if they existed directly before the patient's eyes, and they vary in structure from the most complex to relatively simple kinds.

GENERAL CONSIDERATIONS

While we have used the term hallucination to describe the order of visual phenomena under consideration, we do so knowing there are many objections to this term. The word hallucination means different things to various persons. There are some writers who think of the word according to Esquirol's⁴⁵ definition: "An internal conviction of a sensation without an external stimulus." The phrase "internal conviction" implies a loss of the sense of reality, an absence of judgment concerning the relation of the image to the world about the experimenter. This definition is almost tantamount to declaring the victim of such an experience psychotic, as indeed was probably intended. However, as we now know, some definitely psychotic persons are aware that their visual experiences are truly imaginary. Furthermore, otherwise normal persons may have for one reason or another visions so vivid and detailed that they have every reason to accept the validity of their experience.⁵⁰ This definition obviously cannot be accepted for the type of visual disturbance with which we are dealing. Nor can we drop the phrase "internal conviction" and speak of hallucination as "a perception without an external stimulus." No sensation can arise without a stimulus no matter how elusive it may be. As we have shown, it may not be obvious to the observer, because the stimulus may lie within the head of the patient.

The real difficulty is that language lacks a precise word to describe visual images arising from an abnormal stimulus of the visual apparatus in the presence of a normal and critical intelligence. In speaking of general sensory function, the term dysesthesia is used, which refers to those sometimes complex sensations arising from abnormal excitations of the sensory pathways. The visual phenomena with which we are dealing are exactly comparable to the dysesthesias in the cutaneous and kinesthetic sensory spheres. For lack of a better word, the term hallucinations has to cover both the percepts due to abnormal excitations of the neural parts of the visual pathways and those of an entirely different origin, arising as psychic projections as in certain psychoses, or for that matter the images experienced by eidetic persons or those seen in the hypnogogic state.

Therefore, one must either broaden the conception of the word or adopt a noncommittal definition, such as Dorland's,⁵¹ which avoids entirely the problem of pathogenesis. Thus hallucination "is the perception of an object or phenomenon which has no external existence."

50. McDougall, W.: Hallucination, in *Encyclopaedia Britannica*, ed. 14, New York, Encyclopaedia Britannica, Inc., 1929, vol. 11, p. 105.

51. Dorland, W. A. N.: *The American Illustrated Medical Dictionary*, ed. 16, Philadelphia, W. B. Saunders Company, 1932.

We prefer to use this definition in this sense, since the word hallucination has been traditionally used to describe the visual phenomena occurring in the course of tumors of the brain.

The term hallucination has also unfortunately been used to describe phenomena which are clearly illusory. An illusion is a misinterpretation of something seen! It is the superimposition and fusion of cognitive and affective elements on and with optical sensations reaching consciousness through a normal visuoneural apparatus. The misinterpretation may arise either from a defect in the optical or muscular apparatus of the eye which causes a distorted or perverted image or from a general disturbance of consciousness which misidentifies and confuses the sensory presentations. Both may operate under certain circumstances. In the illusions, falsely called hallucinations, the retina, optic nerves and projection fibers are all anatomically and functionally normal.

Hansel⁵² (1906) pointed out that persons with myopia may have an inaccurate impression of size, distance and color. Much detail is therefore left to the imagination, and almost any object can be imagined as having been seen. Corneal opacities may delude the patient into believing that he sees clouds or smoke. Opacities of the vitreous may be imagined as flies or webs which elude the patient's grasp. The more highly imaginative the person, the more complicated will be the image. There is a second variety of illusions, often reported as hallucinations, in which the error depends on a clouded consciousness. In the familiar delirium or in the organic encephalopathies due to whatever cause the environment may be misinterpreted, so that a waving or moving object is seen as a snake, or spots on the pillow are thought to be insects.

Morel⁵³ (1933) has pointed out that the patient with delirium tremens has a positive scotoma which is the source of the illusion. When the patient is sober it can be mapped out with the perimeter, and the patient is subjectively conscious of a slightly foggy area in his field of vision. With the clouding of consciousness and loss of judgment incident to intoxication, the foggy areas become small animals, objects, bugs, etc. Schroeder⁵⁴ (1921), Terson⁴¹ (1930), Lhermitte and de Ajuriaguerra⁵⁵ (1936) all came to the conclusion, though unfortunately using the term hallucination, that phantopsias following disease of the eye are really profound misinterpretations on the basis of a general disturbance of the brain. In this group also fall the visions of the

52. Hansel, cited by Posey and Spiller.³⁸

53. Morel, F.: Positive Scotoma and Visual Hallucinations, *Rev. d'oto-neuro-opt.* **11**:81, 1933.

54. Schroeder, P.: Hallucinoses and Hallucinations, *Monatschr. f. Psychiat. u. Neurol.* **49**:189, 1921.

55. Lhermitte, J., and de Ajuriaguerra, J.: Visual Hallucinations and Lesions of the Visual Apparatus, *Ann. méd.-psychol.* **94**:321, 1936.

hashish smoker or mescal drinker. Marshall ⁵⁶ (1937), in an astute analysis, pointed out that the visions seen during mescal intoxication are illusory perceptions of the choriocapillary circulation. “. . . from such various shadowy forms in a state of mind given to phantasy, innumerable compositions are possible.”

In all these examples one sees disturbances in apperceptual function comparable in the general sensory sphere to so-called haptic hallucinations. These various illusory experiences are obviously different in origin from the phenomena existing in the cases we have described. In these, there is the perception of visual excitations arising from non-physiologic stimulation of the neural portions of the optic system occurring in persons with intact minds and personalities. All of them possessed critique and judgment and realized that their visual experiences were abnormal and hallucinatory.

PATHOGENESIS AND PSYCHOPATHOLOGY

Much of the confusion respecting visual perception and visual hallucinations revolves about a particular attitude toward vision, in that it is thought of as a special sense or one of the special senses. Because of this point of view, it is considered apart from the general sensory systems. A new point of view sometimes serves to make clear problems that seem confused and to link phenomena seemingly disconnected into a generalization. It is submitted that vision and its anatomic correlate, the opticovisual pathways, are analogous in every respect to the general sensory systems and that visual dysfunctions are comparable in both the physiologic and the psychologic sphere to those of the general sensory apparatus.

Wilson ⁵⁷ (1927), in a penetrating and closely reasoned argument, pointed out that dysesthesias, such as the feeling of wind blowing on the skin, ants crawling on the skin and other complex sensory perceptions, do not, because of their complexity of character, necessarily arise either from cortical irritation or from lesions of the thalamus. He clearly demonstrated that such perceptions can and do arise from lesions at every physiologic level of the sensory pathways. This implies, of course, that such sensations can be excited by irritation of any portion of the sensory fiber or any one of the intercalated neurons from the peripheral nerves to the cortex. More recently, Davison and Schick ⁵⁸

56. Marshall, C. R.: *An Inquiry into the Causes of Mescal Visions*, *J. Neurol. & Psychopath.* **17**:289, 1937.

57. Wilson, S. A. K.: *Dysesthesiae and Their Neural Correllates*, *Brain* **50**:429, 1927.

58. Davison, C., and Schick, W.: *Spontaneous Pain and Other Subjective Sensory Disturbances: A Clinicopathologic Study*, *Arch. Neurol. & Psychiat.* **34**:1204 (Dec.) 1935.

(1935) published reports of a number of cases in which complex sensations arose from lesions at all levels of the sensory system. They cited a large number of cases from the literature, pointing to the fact that complicated dysesthesias may arise from lesions of the peripheral nerve, spinal cord, bulb, pons, thalamus and cortex. Though vision is a different type of perception, it by no means follows that it is a special kind of perception. There can be little doubt but that there exists a biologic and physiologic unity of all types of sensory perception. Laws of general applicability to cutaneous and kinesthetic sensation are applicable to vision. There is no evidence we know of in contradiction to this point of view.

Excitations arising in the tactile or kinesthetic sphere, like visual sensations, emerge into a perceiving consciousness and are modified by attention, interest, past memories, emotional associations and a number of other elements of the psychic series. The consequent sensation or vision that the patient believes he feels or sees is the product of a highly complicated psychologic synthesis. In what manner these higher mental integrations are accomplished is largely unknown and is a problem for experimental psychology. We shall attempt to point out the nature of some of the possible factors in a later paragraph.

There seems to be nothing strange or unexpected in the occurrence of visual images resulting from irritation and stimulation of these pathways at any point from the retina to the brain. The excitations are conveyed to the brain, where the purely sensory presentations are fused with the psychic factors mentioned and experienced as a visual perception. Since experience determines the projection of perceptions, visual perceptions are projected as if they came from the external world. Thus it does not matter that the person may be blind; he nevertheless "sees" the images as the result of the abnormal visual stimulation. The same thing exactly occurs in the dysesthesias which may be experienced in a totally anesthetic area. It is apparent, however, that in spite of the loss of vision resulting from destruction of the visual fibers, enough viable fibers must be present to carry the excitations to the higher integrative mechanisms, because otherwise they could not appear in consciousness.

We believe that all the visual hallucinations described as associated with tumor of the brain or with other focal lesions of the neuro-optic apparatus arise in this way, regardless of whether the lesion affects the retina, optic nerve, optic tracts, projection fibers or visual cortex. The psychologic mechanisms that transform sensory presentations into perceptions and endow these perceptions with meaning do not lie only in the visual cortex but represent the total integrative activity of the psyche.

Once one considers the phenomenon of vision above the physiologic level, tremendously complicated factors are found operating. How simple excitations are fused with memory, experience and affectual ingredients and how they are modified by attention and interest, finally to be perceived as an image endowed with specific character, are largely unknown and defy physical analysis.

The crux of the problem, however, is why the same lesion in one person provokes relatively simple visual experiences and in another provokes complicated visions. As long as it was believed that this depended on the stimulation of more or less "psychically organized" cerebral centers, it followed that visual hallucinations were central phenomena. But when this is shown not to be the case, some other explanation is demanded. The principles of cerebral localization cannot be applied to explain highly complex intellectual syntheses, which require on the face of it the integration of the entire brain. Morton Prince⁵⁹ (1923) has summed it up succinctly: ". . . it is incredible that such complex phenomena should be correlated with any particular focus in the brain. I do not think it possible that irritation of any particular focal area can produce such a complicated psychologic phenomenon as an hallucination."

One can attempt to understand, however, the difference between the images experienced by a consideration of the image-making functions of the mind and the variability of this function among persons. The phenomenon of eidetic imagery contributes to span the gap between the purely physical and the purely psychic. Many years ago Galton⁶⁰ (1883) in a study of visual imagery noted that the visual memory of persons varied greatly. To some, visual imagery had no meaning, while others could revive an earlier visual experience with hallucinatory clarity. The latter persons he called visualizers. Jaensch and his school restudied this subject carefully and made the observation that all children possessed the ability actually to "see" their visual memories and that in some persons this faculty persisted into adult life. For these persons whom he called "eidetics," a recalled memory image is "seen" before the eyes of the subject as if it possesses objective reality. The eidetic image possesses spatial localization, it is highly detailed and is subject to voluntary recall. Between the eidetic and the nonvisualizer are a whole series of gradations in which the image can be recalled with more or less clarity. It is this constitutional factor of natural imagery which, in the words of Miller⁶¹ (1931), ". . . contributes an undertone to hallucinosis. It

59. Prince, M., in discussion on Horrax.²⁸

60. Galton, F.: *Inquiries into Human Faculty and Its Development*, London, Macmillan & Co., 1883.

61. Miller, E.: *The Affective Nature of Illusion and Hallucination: Eidetic Imagery*, *J. Neurol. & Psychiat.* **12**:1, 1931.

is the image-making functions of the mind that gives meaning to percepts if it does not actually manufacture them. The eidetic image is the biological process which links external reality to the neural mechanisms and to the world of neural forms."

For persons having a high degree of visual imagery, the sensory presentations produced by the abnormal stimulation of the neuro-optic apparatus, such as occurs in association with irritations of the optic nerves, are projected as rich and elaborate images. For those with a poor degree of visual imagery or absence of it, the visual images are simple and relatively "crude." Other factors of the psychic series may modify the productions of the image-making function. The affectual tensions, the degree of intelligence, the alertness of the patient and even deep subjective ingredients may in subtle ways condition the character of the final image. Visual memory and previous visual experience are the *sine qua non* of any visual hallucination. It is well known that congenitally blind children do not have visual hallucinations, even in delirium, although they have auditory and kinesthetic hallucinations (Tuke,⁶² 1888; Ormond,³² 1925). We do not know whether the experiment has ever been made, but we doubt whether the simplest type of visual image can be evoked by electrical stimulation of the visual cortex of a congenitally blind child. These considerations led Pear⁶³ (1927) to speak of a hallucination as "the percept of a memory."

Analogous image functions are seen in other sensory fields than the visual. Playing the piano "by ear" and reproducing complicated musical passages after one hearing or the ability of some persons to find their way through a complex maze blindfolded, after but one trial, hints at the presence of auditory and kinesthetic phenomena akin to the position of eidetic imagery in the visual sphere. It is highly probable that the same kind of psychologic activity underlies the experience of those suffering from cutaneous dysesthesias. With the same lesion, one patient may feel only numbness or tingling, while another feels water trickling or ants crawling on the skin. These more elaborate perceptions are not "imaginary," for the patient is equally aware that they have no reason for existence. Prince⁶⁴ (1922) stated that "irritation produces simple sensory phenomena, but the possibility of irritating factors becoming the excitants of organized complexes of neurons underlying hallucinations cannot be excluded."

62. Tuke, D. H.: Hallucinations and the Subjective Sensations of the Sane, *Brain* **11**:441, 1888.

63. Pear, T. H.: Recent Investigations on Visual Imagery with Special Reference to Hallucinations, *J. Ment. Sc.* **73**:195, 1927.

64. Prince, M.: The Experimental Study of the Mechanisms of Hallucinations, *J. Nerv. & Ment. Dis.* **56**:248, 1922.

We see in the psychologic as well as in the physiologic field the same integrating and synthesizing activities which establish the unity in many ways of all types of sensory perception. It seems hard to believe that abnormal perceptions of vision can be produced only by cortical irritation, while abnormal perceptions in the cutaneous or kinesthetic spheres can arise from irritations at any level in the sensory system. Even in the other so-called "special senses" the same generalization holds. Auditory hallucinations have been reported in association with lesions of the peripheral apparatus (Colman,⁶⁵ 1894; Goldstein,⁶⁶ 1908; Klieneberger,⁶⁷ 1912, and Rhein,⁶⁸ 1913), and we have several cases in our records in which tumors compressing the olfactory nerves in the olfactory groove have produced typical olfactory hallucinations.

These not too rare cases substantiate the opinion that the physiologic level of the stimulation is relatively unimportant; it is the psychologic image-making function of the mind which clothes the excitations and projects them as more or less elaborated perceptions.

The clinical explanation of the relative frequency of visual hallucinations in hemispheric lesions as opposed to the infrequency of their occurrence in association with lesions of the optic nerves is a simple example of incidence. Primary and metastatic tumors involving the visual cortex and subcortical visual radiations are more common than tumors involving the optic nerves. The same is true for vascular lesions. In migraine, also, the cortex is presumably the site of the morbid process. This is again so with the epilepsies. Cerebral trauma, birth injuries and a number of other processes produce lesions almost entirely in the hemispheres where they are in the position to affect the visual cortex or subcortical fibers. Moreover, the visual cortex and projection pathways within the brain occupy large areas, allowing the possibility of irritation from many points.

Our 16 cases of visual hallucinations were culled from 139 cases of verified adenoma of the pituitary body and from 84 cases of tumor of the hypophyseal duct. A number of cases were rejected because there were obvious mental disturbances, and one could not be certain whether the hallucinations were due to the patients' misinterpretations of their surroundings (illusory). Several histories stated that hallucinations were present but gave no further information. If one includes the latter, there were 22 cases of visual hallucinations among 139 cases of adenoma,

65. Colman, W. S.: Hallucinations in the Sane Associated with Local Organic Disease of the Sensory Organ, *Brit. M. J.* **1**:1015, 1894.

66. Goldstein, K.: Zur Theorie der Hallucinationen, *Arch. f. Psychiat.* 584, 1908.

67. Klieneberger, O.: Gehörtäuschungen bei Ohrenerkrankungen, *Allg. Ztschr. f. Psychiat.* **69**:285, 1912.

68. Rhein, J. H.: Hallucinations of Hearing and Disease of the Ear, New York M. J. **97**:1236, 1913.

of which 12 are presented here. There were 6 cases of visual hallucinations among the 84 cases of tumor of the hypophysial duct, there being sufficient information concerning 4 to warrant description. Since the great bulk of the latter tumors occur in children who do not give their histories and are largely incapable of reporting such experiences, the frequency of visual hallucinations appears low; if these tumors occurred in adults, possibly a greater number of hallucinations would be reported.

RELATION OF HALLUCINATIONS TO HEMIANOPIA

It was pointed out earlier that it is generally believed that visual hallucinations occur in the hemianopic field. This fact is used for the clinical localization of lesions. This notion has become fairly well fixed, even though there have been a large number of cases in which this has not occurred. Craig⁶⁹ (1836), Westphal¹⁵ (1881), Ingels,¹⁶ (1882), Bennett and Gould³⁴ (1887), Henschen¹⁴ (1890), Déjerine, Sollier and Auscher¹⁷ (1890), Sittig⁷⁰ (1925), Horrax²⁸ (1923), Holmes⁷¹ (1931), Johnson (1933), Stone⁷² (1934), Spiller⁷³ (1934) and Riddoch⁷⁴ (1935) have all recorded cases in which the hallucinations appeared either in the entire field or in the intact field. This has been also true in cases of hallucinations seen in association with migraine, in which the images have not appeared in the hemianopic field (de Schweinitz,³¹ 1889, and Ormond,³² 1925). One should mention the fact that in a number of cases in the literature the hallucinations have occurred in the absence of any defects in the visual fields. (Horrax,²⁹ 1923, and Allen,⁷⁵ 1930, among others). In Horrax' cases three fourths of the patients with visual hallucinations had no defects in their fields on admission to the hospital. In Sanford and Blair's⁷⁶ series of 22 cases of hallucinations occurring in association with tumors of the temporal lobe, the defects were localized to the hemianopic field in only 2 instances.

69. Craig, J.: History of a Case of Spectral Illusions with Subsequent Loss of Memory of Words and Notes with the Appearance on Dissection, *Edinburgh M. J.* **46**:334, 1836.

70. Sittig, O.: A Clinical Study of Sensory Jacksonian Fits, *Brain* **48**:233, 1925.

71. Holmes, G.: A Contribution to the Cortical Representation of Vision, *Brain* **54**:470, 1931.

72. Stone, L.: Paradoxical Symptoms in a Right Temporal Lobe Tumor, *J. Nerv. & Ment. Dis.* **79**:1, 1934.

73. Spiller, W., cited by Stone.⁷²

74. Riddoch, G.: Visual Disorientation in Homonymous Half Fields, *Brain* **58**:376, 1935.

75. Allen, I. M.: A Clinical Study of Tumors Involving the Occipital Lobe, *Brain* **53**:194, 1930.

76. Sanford, H. S., and Bair, H. L.: Visual Disturbances Associated with Tumors of the Temporal Lobe, *Arch. Neurol. & Psychiat.* **42**:21 (July) 1939.

In our cases there was no constant relation between the portions of the apparent field in which the images were seen and the areas in which vision was lost. Every possible combination was represented. In cases 1, 2, 3, 4, 5, 7, 8, 9, 12 and 13 the defects of the visual fields varied widely. In case 3 only a relative scotoma was present in the right eye, while in case 13 only small nasal islands of vision were retained. In these cases the hallucinations were seen as if the objects existed in front of the patients. In cases 6, 10, 14 and 15 the hallucinations were lateralized, although the field defects ranged from homonymous hemianopia to complete blindness. Only in case 10 did the hallucinations occur in the hemianopic field. In case 11 the images were seen only in front of the blind left eye. This was the single case in which clearly unilateral hallucinations occurred. The occurrence of visual hallucinations in the totally blind demonstrates that the visual experiences are not illusory, for there is nothing seen to be misinterpreted. Moreover, when the hallucinations occurred the patients were aware that the images were not related to objective reality. They retained their critique and were distressed by the unwanted and interfering images. In 1845 and 1846 there appeared two remarkable papers written by Abell,⁷⁷ giving an account of his own experience on going blind. In 1841 vision began to fail in his right eye; this was accompanied by smokelike visions during the day. At night his room would appear to be brightly illuminated, and he saw beautiful patterns on the walls. Vision then began to fail in his left eye, and ". . . the sight became so distorted as to cross the axis of vision." In four years he became totally blind "without suffering pain or inflammation." From this time on he began to experience the most remarkable visions of vivid and unfamiliar landscapes, parades of gorgeously caparisoned soldiers, buildings, cities and multitudes of persons of all sexes, animals and children and many other wonderful and strange sights. He stated: "The darkest nights were no obstacle to my seeing these creatures, for my room was always sufficiently light to discern every feature of their faces. . . . I saw more people and more beautiful works of art than I had ever seen before. My greatest suffering has been from the luminous or lighted-up appearance in my eyes which prevented me from sleeping." The images always passed before his eyes moving from right to left. From Abell's account it appears likely that he may have suffered from a tumor involving his optic nerves. More recently, such cases have been reported by Terson⁴¹ (1930) and Trillot and Carlet Soulages⁷⁸ (1937).

77. Abell, T.: Remarkable Case of Illusive Vision, Boston M. & S. J. **33**:409, 1845; Optical Illusions, *ibid.* **34**:52, 1846.

78. Trillot, J., and Carlet Soulages: Late Development of Differentiated Visual Hallucinations in a Woman Blind for More Than Twenty Years, *Ann. méd.-psychol.* (pt. 2) **95**:109, 1937.

In our 3 cases (6, 15 and 16) images were also seen though the patients were totally blind. In case 15, during the year following operation the patient was perfectly clear and aware that the visions she was seeing were unreal. Later the onset of mental clouding makes analysis difficult, but it seems fair to believe that in spite of her obtundity the stimulus for the hallucinations was still in the compressed and irritated optic nerves.

THE ORGANIZATION OF THE HALLUCINATION AND ITS RELATION
TO THE SITE OF LESION

The greatest importance for localizing value has been placed on the distinction between simple or crude visual hallucinations and the complex or elaborate variety. This attitude again stems from the concept that hallucinations occurring in association with focal lesions are central phenomena and that the difference in organization of the visual image depends on the psychic organization of the visual area irritated. Aside from the objections to this belief already stated, it may further be objected to on the grounds that clinically this is not always the case and, secondly, on psychologic grounds, that no distinctions are possible between so-called simple and so-called complex hallucinations.

Craig⁶⁹ (1846), Ingels¹⁰ (1882), Reinhard²² (1887), Putzel⁷⁹ (1888), Henschen¹⁴ (1890), Seguin²¹ (1886), Wollenberg¹⁸ (1890), Harris⁸⁰ (1897), Climenko⁸¹ (1915), Horrax and Putnam²⁹ (1932) and Sanford and Bair⁷⁶ (1939) recorded cases of complex, formed hallucinations of persons, objects, animals, etc., in association with lesions of the occipital lobe. On the other hand, Cushing²⁷ (1921), Horrax²⁹ (1923) and Sanford and Bair⁷⁶ (1939) recorded so-called unformed hallucinations in their series of cases of tumor of the temporal lobe. The latter authors found crude images in 11 of 22 cases of hallucination occurring in association with tumors of the temporal lobe. It appears, therefore, that when the cases in the literature are scrutinized, the categoric opinions that distinctive types of hallucinations are peculiar to either the temporal or the occipital lobe become highly doubtful. As is well known, the number of tumors restricted to the occipital lobe are relatively uncommon compared to those of the temporal lobe. Thromboses of the posterior cerebral artery are also infrequent. These facts limit the number of lesions of the occipital lobe that may be studied to a rela-

79. Putzel, L.: A Case of Cortical Hemianopia, with Autopsy, New York M. Rec. **33**:599, 1888.

80. Harris, W.: Hemianopia with Especial Reference to Its Transient Varieties, Brain **20**:308, 1897.

81. Climenko, H.: Case of Tumor of the Right Occipital Lobe, J. Nerv. & Ment. Dis. **43**:826, 1915.

tively small number. The study of an increased number of lesions of the occipital lobe may well alter our clinical impressions.

The distinction has been made between the composition of a crude, or simple, hallucination and the composition of a complex, or formed, hallucination without the difference being entirely clear. The two have been thought of as comprising separate order of phenomena, but this attitude is not compatible with psychologic facts. Duke-Elder⁸² (1932) stated: "It is impossible to experience a pure isolated sensation—even the simplest and most elementary perceptual process comprises a synthesis of many different impressions." A moment's reflection convinces one that there is nothing crude about the images of "leaping flames," "colored stars," "floating bubbles" or even "wavy black lines." They possess color, often form, sometimes motion, and frequently direction of movement. Moreover, they are spatialized, some seeming to occur at a distance and some nearby, or they are localized to a particular portion of the field. They are recognized by the patient as being a "thing" just as the more elaborate images are. Regardless of whether an image is a simple "thing," it none the less possesses enough characteristics to be recognizable and namable. To dismiss these types of images by calling them photomas is doing violence to the facts and begging the question of their significance. When such images occur under other circumstances, as in association with known schizophrenia, for instance, they are given full value as a hallucination, both as to content and as to meaning.

Between the simplest type of visual image and the most elaborate there are imperceptible gradations, so that even if one adopted a sharp distinction between the extremes one would find it difficult to appraise some of the intermediate examples in terms of only "simple" or of only "complex."

Some of our patients saw "floating colored bubbles" and "colored balls"; by some the bubbles were seen to burst. Others saw "rockets going off in all directions." One saw geometric patterns like a kaleidoscope and numerals. One patient saw spinning wheels; another saw colored stars against a differently colored background. These images are indeed complicated, and it would be quarreling with logic to call them crude. Even in those cases in which faces and persons were seen, other and less complicated images, like patterns, colors and flashes of light, were frequently seen along with them.

From this point of view there are really no crude or complex images but merely hallucinations of different things. As we have indicated

82. Duke-Elder, W. S.: *Textbook of Ophthalmology*, St. Louis, C. V. Mosby Company, 1932, vol. 1.

before, the things which a patient sees as well as the complexity of them are dependent on the syntheses which the psyche makes with the excitations presented to consciousness. It seems to matter but little at what point in the neuro-optic apparatus the stimulus is given. The final perception does not correspond to the immediate stimulus but conforms to the background provided by experience.

In conclusion, it is only fair to state that we know little concerning the personality structure of the 16 patients whose case histories are presented in this study. In 4 cases (5, 11, 12 and 16) it was possible to interview and study the patients at some length. In none of these were schizoid or hysterical elements obvious. None of them had had previous hallucinatory experiences. In several other cases in which information was obtained through correspondence with the patient and his family, no history of previous hallucinatory episodes was elicited. In no instance did the hospital record state that any of the patients had had hallucinations under previous circumstances. In view of this evidence it seems unlikely that we are dealing with persons with latent psychoses. Furthermore, in several cases the hallucinations were no longer experienced after removal or partial removal of the tumor and freeing of the optic nerves. In all instances the patients discussed their hallucinations freely and objectively—a rather rare occurrence in the psychoses. The content of the hallucinations did not in general possess material of emotional significance to the patients.

SUMMARY AND CONCLUSIONS

It was pointed out that in view of the historical development of the principles of cerebral localization and the observations that visual hallucinations occurred in association with lesions of the temporal and occipital lobes, certain concepts developed respecting the clinical significance and pathogenesis of visual hallucinations associated with focal cerebral lesions. Clinically, it came to be believed (1) that visual hallucinations are central irritative phenomena, (2) that visual hallucinations occur only in the homonymous hemianopic fields or in the field contralateral to the lesion and (3) that formed hallucinations indicate a lesion in the temporal lobe while unformed hallucinations signify a localization in the occipital lobe.

From a pathogenetic point of view, the complexity of the image was thought to be dependent on the irritation of either highly or lowly psychically organized cortical centers, the ones more psychically organized yielding on stimulation elaborate images and the ones less organized yielding crude images. It followed according to these concepts that lesions of the peripheral portions of the neuro-optic apparatus could not provoke hallucinations.

Although these ideas appear to be widely believed and are so expressed in the current textbooks, a perusal of the literature indicates not only that there have been objection to these conceptions but that cases are on record clearly demonstrating that visual hallucinations may arise from lesions of the retinas, optic nerves, optic tracts and visual projection pathways.

Sixteen cases of visual hallucinations occurring in association with tumors compressing the optic nerves and chiasm are reported. The visual phenomena are analyzed in respect to the organization of the images and to the relation between the portions of the field in which the hallucinations are subjectively experienced and the portions of the fields which are objectively blind.

It was found that all types of visual hallucinations from the simplest to the most elaborate occurred in these cases. A review of the literature on lesions of the temporal and occipital lobes also shows that there has been no constant correspondence between the site of the lesion and the character of the hallucination. Furthermore, it was demonstrated that no constant relation existed between the portions of the field in which the images were subjectively projected and the areas of objective loss of vision. This fact, too, has been noted in cases of hallucinations occurring in association with lesions of the temporal and occipital lobes.

Since visual hallucinations are possible in association with lesions at any level of the neuro-optic apparatus, it follows that they are not necessarily central phenomena provoked only by irritation of the visuo-sensory or visuopsychic cortex. They represent highly complex psychologic syntheses, which are set in action, so to speak, by sensory excitations arising from any part of the neural apparatus of the visual system. They are the result of the total integrative activities of the mind, which fuse memory, affectual associations and previous visual experience with simple sensory presentations to create an image. They are modifiable by interest, attention and the degree of intellectual endowment.

One of the most important factors determining the final, if not the basic, character of the image is a constitutional one, the native tendency toward visual imagery. Those in whom it is poorly developed experience the more simply constructed visual hallucinations, while in those highly endowed, the images are elaborate.

In all of our cases the images arose through the nonphysiologic stimulation of the neural portions of the visual apparatus. They were, and this is an important distinction, perceived within the structure of an intact intelligence and personality. Though language lacks a precise word to describe images arising under these circumstances, the term hallucination has been used out of deference to custom. The point

was discussed that unfortunately the term hallucination is also used to describe visual images arising under totally different circumstances as well as to describe images of a different order, which analysis shows to be illusory.

The mechanism by which hallucinations are produced in consequence of peripheral lesions of the neuro-optic apparatus is exactly comparable to that operating in the production of dysesthesias in the general sensory sphere. The often complicated dysesthesias may appear in association with lesions of the sensory system at every physiologic level. In a general way, all sensory systems show a physiologic unity and all sensory perceptions a psychologic unity.

The conclusions reached by the study of the cases in the literature and by the analysis of the cases here reported are: 1. Visual hallucinations in themselves have no localizing value whatever in focal diagnosis. 2. Visual hallucinations may be provoked by lesions at any level of the neuro-optic apparatus. 3. Visual hallucinations are not due to local cortical excitability but are psychologic phenomena, involving the total integrative activities of the mind. 4. The complexity of the images depends on psychologic and constitutional factors and not on cortical psychic organization. 5. There is no constant relation between the portion of the field into which the hallucinations are projected and the objectively blind areas.

News and Notes

EDITED BY W. L. BENEDICT

SOCIETY NEWS

The American Academy of Ophthalmology and Otolaryngology.—Dr. Frank R. Spencer, of Boulder, Colo., was chosen president-elect of the American Academy of Ophthalmology and Otolaryngology at the annual session held in Chicago on October 11. He will succeed Dr. Frank E. Brawley, of Chicago, when the latter becomes president of the academy on January 1. Dr. Spencer is a graduate of the University of Michigan Medical School and has been a member of the faculty of the University of Colorado School of Medicine since 1905.

The academy decided to act as sponsor for a proposed Pan American Congress of Ophthalmology and Otolaryngology. South American physicians attending the meeting in Chicago will arrange for the attendance of delegates from their respective countries to such a congress to be held in connection with the next meeting of the academy. It is understood that invitations to each of the countries concerned will have the sanction of the Department of State and will be forwarded through diplomatic channels.

Other officers elected were Drs. Arthur W. Proetz, St. Louis, first vice president; Joseph F. Duane, Peoria, Ill., second vice president, and Charles T. Porter, Boston, third vice president; Secord H. Large, Cleveland, comptroller, and William P. Wherry, Omaha, executive secretary, reelected. Dr. Erling W. Hansen, Minneapolis, was elected secretary for public relations, succeeding Dr. Ralph A. Fenton, Portland, Ore., who resigned. The following secretaries were reelected: Drs. William L. Benedict, Rochester, Minn., for ophthalmology; John L. Myers, Kansas City, Mo., for otolaryngology; Dean M. Lierle, Iowa City, for instruction in otolaryngology, and Albert D. Ruedemann, Cleveland, for instruction in ophthalmology. Dr. Albert C. Snell, Rochester, N. Y., was elected a member of the academy's governing council, and Dr. Frederick C. Cordes, San Francisco, to represent the academy on the American Board of Ophthalmology.

The academy continued the following appropriations for research: Dr. Olof Larsell, University of Oregon Medical School, Portland, \$400 for research on development of the internal ear; Dr. M. H. Lurie, Harvard Medical School, Boston, \$400 for research on the balancing apparatus of the ear, and for the Army Medical Museum at Washington, D. C., \$1,500 for maintaining collections of pathologic specimens in diseases of the eye, ear, nose and throat. An appropriation of \$1,500 was also made for the establishment under the supervision of the academy of reading courses for young physicians serving as residents in hospitals who are preparing for specialization in diseases of the eye, ear, nose and throat. A grant of \$400 was made to Dr. Spencer for research on the action of drugs on tubercle bacilli in the nose and throat. The committee on physiologic optics received \$50 and the committee on orthoptics \$200.

The 1940 meeting will be held in Cleveland, October 6 to 11, with headquarters in the Hotel Cleveland.

Association for Research in Ophthalmology.—The trustees of the Association for Research in Ophthalmology announce that at the

1940 meeting a cash prize of \$100 will be offered for the paper which in their judgment shows most originality and exemplifies best the spirit of research in ophthalmology.

GENERAL NEWS

Residency in Ophthalmology, St. Luke's Hospital, New York.—St. Luke's Hospital, New York, announces an eighteen month residency in ophthalmology and the discontinuance of its former combined residency in ophthalmology and otolaryngology. The service will commence on January 1, annually. For the first six months the appointee will be enabled, through the hospital's affiliation, to take the full time course of instruction in basic sciences and principles of ophthalmology given at the Graduate School of the College of Physicians and Surgeons of Columbia University. During the entire residency of eighteen months, accommodation will be provided in the hospital. A small monetary allowance will be paid during the clinical portion of the service.

On the termination of the hospital service, the graduating resident will be assisted in securing a clinical appointment to complete the requirements for the American Board of Ophthalmology.

This residency offers an active ward, clinic and private ophthalmologic service, with the reciprocal consulting facilities in all departments of medicine which only a large general hospital can provide.

PERSONAL

Prof. Anton Elschmig (1863-1939).—Prof. Anton Elschmig died in Vienna, on Nov. 13, 1939, following an automobile accident. Professor Elschmig was 76 years old. He retired to Marienbad, Czechoslovakia, from the German University in Prague in 1934.

A special article written by Dr. Bernard Samuels in commemoration of Professor Elschmig's seventieth birthday appeared in the July 1933 issue of the ARCHIVES, page 110.

Dr. Robert Blue (1876-1939).—Dr. Robert Blue, assistant professor of ophthalmology at Northwestern University and attending ophthalmologist at the Wesley Memorial Hospital in Chicago, died suddenly on Dec. 8, 1939, of cardiac failure.

Notices

Cumulated Index of the Archives of Ophthalmology.—Requests have been received for a ten year index of the ARCHIVES OF OPHTHALMOLOGY. Before serious consideration is given to the production of a cumulated index, it is desirable to know whether the demand for it would be sufficient to warrant its sale at not to exceed \$5 a copy; that is, whether one thousand copies could be sold. It will be appreciated if those who are interested in such an index will fill out and send the form which appears below to the Managing Editor at the publication office, 535 North Dearborn Street, Chicago.

I SHOULD BE WILLING TO SUBSCRIBE TO A CUMULATED INDEX OF THE ARCHIVES OF OPHTHALMOLOGY AT \$5.00.

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Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

HISTOLOGIC APPEARANCE OF THE CHORIOCAPILLARY LAYER OF THE CHOROID. A. HUDELO, *Ann. d'ocul.* 176: 186 (March) 1939.

The author expresses the opinion that the capillary layer of the chorio-capillaris is as much a histologic unit as the free lamina, as may be proved by detachment. This is interesting, because certain authors have written opposing the division of the choroid into five layers. However, from a study of his photomicrographs and of sections from which they were made, the author concludes that as regards the close detail it is necessary to side with the opinion of Wolfrum, and that the opinion of Venneman cannot be retained. The dimensions are those which Wolfrum gave for the capillaries of the posterior pole; the walls are formed of endothelial cells, and the surrounding interlacing fibers are not adherent. On the contrary, Wolfrum's opinion that the nuclei are situated on the external surface of the capillary is not acceptable to the author, for he observed them occupying the sides and not the scleral surface of the capillaries.

S. H. MCKEE.

Biochemistry

THE CALCIUM CONTENT OF THE AQUEOUS, LENS AND BLOOD IN RABBITS AFTER THE ADMINISTRATION OF PARATHYROID EXTRACT. A. TALIERCO, *Ann. di ottal. e clin. ocul.* 66: 925 (Dec.) 1938.

The author removed one eye from each of a series of rabbits, at the same time withdrawing blood from the vein in the ear. The calcium content of this material was determined, after which parathyroid extract was given for twenty to ninety days, when the other eye and a second sample of blood were removed for similar determinations. The dose of parathyroid extract employed was 10 to 20 Collip units daily. Eleven animals were studied. An increase in calcium was constantly present in the blood and aqueous after the administration of the parathyroid extract, but no change in that of the lens was noted. This seems to indicate that an increase of calcium in the blood and aqueous produces a decreased permeability of the lens capsule for calcium. The reverse process has been shown to occur after parathyroidectomy, when a decrease in the calcium of blood and aqueous accompanied by increased permeability of the lens capsule, as indicated by the accumulation of calcium in the lens, was noted.

S. R. GIFFORD.

Color Sense

"SIMPLE UNCOMPLICATED" FORM OF CONGENITAL TOTAL COLOR BLINDNESS: REPORT OF A CASE. E. HEINSIUS, *Klin. Monatsbl. f. Augenh.* 101: 489 (Oct.) 1938.

A number of authors distinguish between "complicated" and "simple uncomplicated" congenital color blindness. The complicated form, presenting central amblyopia, photophobia, nystagmus and frequently changes in the macula, appears to be the most frequent. Heinsius reports the case of a youth aged 18 who had the simple uncomplicated form of congenital total color blindness unaccompanied by any other symptoms; his vision was perfect. The author concludes that this form is apparently rare and that its origin is probably not to be found in the retina but in a higher sensory center. Observation of the mode of heredity may furnish explanatory data.

K. L. STOLL.

Conjunctiva

TRUE AND FALSE PAPILLARY CONJUNCTIVITIS. E. CORNET, *Ann. d'ocul.* 176: 100 (Feb.) 1939.

The association of papilloma with trachoma was first recorded by May in 1895 and later by Cuénod in 1907; afterward it was recorded by Meyerhof and by numerous oculists. The author states that he has had an opportunity to demonstrate this association on numerous occasions. The conjunctivitis is of true papillary type, there being no question of its being false. Cuénod and Nataf believe that conjunctivitis with small disseminated follicles (Pavia and Dusseldorp), granular syphilitic conjunctivitis (Goldzieher and Sattler) and postmicrobian hyperplastic conjunctivitis all come under the classification gonorrheal conjunctivitis; the hypertrophic metablennorrheic conjunctivitis of Pascheff may belong to the pseudofollicular class. Cornet does not believe that these pseudofollicular formations are real papillomas.

It is certain that the first type constitutes real papillary, or true, conjunctivitis, and the second type, pseudopapillary, or false, conjunctivitis. The first type is divided into three subtypes: (1) papillary conjunctivitis, called spring catarrh; (2) syphilitic papillary conjunctivitis, and (3) secondary papillary conjunctivitis.

The second type is divided into two forms: (1) infiltrated pseudopapillary trachoma and (2) granulopseudopapillary trachoma.

S. H. MCKEE.

STEAM DOUCHE IN THE TREATMENT OF GONORRHEAL OPHTHALMIA. L. G. STEIGELMANN, *Klin. Monatsbl. f. Augenh.* 101: 572 (Oct.) 1938.

Injections of milk and gonococcus vaccine may not show a result before three to four days in cases of gonorrheal conjunctivitis. To prevent sloughing of the cornea during these days, a douche is used which ejects steam at a temperature of 42 C. at a distance of from 10 to 15 cm. It is an apparatus consisting of a kind of thermos bottle connected

with an atomizer. The steam is sprayed on the everted lids after the conjunctival sac is cleansed and epinephrine hydrochloride is instilled. The author observed prompt disappearance of the swelling and decrease of the discharge within two to three days. Analgesic and sedative preparations are added to the steam-forming solution.

K. L. STOLL.

Congenital Anomalies

A DESCRIPTION OF A MONSTER: DIPROSOPUS TETROPTHALMUS.
I. BRODSKY, Brit. J. Ophth. 23:250 (April) 1939.

To the ophthalmologist the features of particular interest pertaining to the monstrosity described here concern the eyes. Three bulgings above two noses contained eyes. The eyes and the palpebral margins of the lateral eyes appeared normal, notwithstanding an epicanthal fold of the inner angle. Inspection of the central bulge removed the impression that there was a single median eye. The palpebral margins provided a clue, in that a cavity was present almost in the middle of the inferior lid, though no ridges or depressions marked the conjunctival surface. Eccentrically placed and slightly to the left of the midline was an iris, approximately equal in size to the lateral irides. Retraction of the eyelids revealed another iris in the upper right quadrant. The corneas over these two irides were continuous, the corneasclerotic junctions taking the form of an hourglass. An illustration shows this well and, in addition, reveals strabismus, the axes of the eyes making an angle of 20 degrees in front.

A block containing the eyes exhibited maximum peripheral fusion at the corneal area. In a sagittal plane a moderately deep annular groove extended around the junction of the eyes and became flattened near the cornea. A common lateral rectus muscle served both eyes, although it was attached principally to the smaller eye, which incidentally received the larger of the two optic nerves. More than half of the cornea of the smaller eye was buried in the fusion, whereas the limbus of the larger eye was just covered.

Transition from sclera to cornea was clearly defined in the larger eye, being demarcated by large vessels. When the sclera was traced back it was found to become thin and compact at the junction of the two eyes. The large vessels at the junction probably indicated the anterior limit of the common sclera; over the front of this the cornea was continuous. Bowman's membrane extended from the extremity of one cornea to the outer edge of the other cornea and crossed the point of junction, where it was less well defined. In the smaller eye, deep to Bowman's membranes, were vessels which did not extend as far as the junction of the two corneas. The substantia propria of the smaller eye lost its lamination at the point where the two corneas were incompletely separated. Descemet's membrane and the endothelium of the smaller eye appeared to end abruptly at the junction of the substantia propria and the sclera. From this point to the angle the anterior chamber was lined by a thin membrane. Posteriorly, the common sclera separated into two parts corresponding to each eye. The vessels of the choroidal coats were large and full, but there was no abnormality of the retina. A wide gap intervened between the two optic nerves.

The article is illustrated.

W. ZENTMAYER.

Cornea and Sclera

EPIDEMIC KERATOCONJUNCTIVITIS OF UNKNOWN ETIOLOGY. R. SCHNEIDER, *Klin. Monatsbl. f. Augenh.* 102:425 (March) 1939.

Schneider reported on a peculiar type of keratoconjunctivitis which he observed in Munich at the meeting of the Bavarian Ophthalmologic Society at Nuremberg on Dec. 4, 1938. At that time he had been suffering from the disease for two weeks. He had noticed an increasing number of cases since August 1938 and had described his observations at the meeting of the Ophthalmologic Society of Munich on Oct. 7, 1938. Oculists and members of their families helped to swell the frequency of this disease since that time, until a decrease was noted about the time of the present report.

In this report Schneider compares the disease with the conjunctivitis resulting from chemical substances, gases or irradiation. In the cases observed symptoms such as hyperemia of the conjunctiva, chemosis and ciliary injection were present, in addition to abrasions and infiltrations of the cornea. The catarrhal swelling increased coincident with the development of the corneal changes, which usually persisted for a longer time. The appearance of the corneal changes varied; however, the epithelial and subepithelial strata were always involved and the lesion frequently extended into the external lamellae of the parenchyma. The lids, aqueous humor, iris and vitreous were involved in some instances. Headache, neuralgia, malaise, parotitis and fever were occasional concomitant symptoms. Only adults were afflicted. Preceding injuries of the cornea and conjunctiva were rarely noted. The usual methods of dyeing and culturing failed to prove the bacterial origin. The causation remained unknown.

Treatment was effective in regard to the symptoms but was ineffective in regard to the course of the disease. The usual duration of the condition was one to two weeks. Recurrences, however, developed, notwithstanding continued treatment, and led to what threatened to become permanent visual lesions. Proof is thus furnished of the comparative severity of this disease.

K. L. STOLL.

EPIDEMIC SUPERFICIAL KERATITIS. SCHULTZE, *Klin. Monatsbl. f. Augenh.* 102:425 (March) 1939.

Schultze, of Nuremberg, reports on the disease described by Schneider in the preceding abstract. Schultze calls the condition keratitis superficialis epidemica. He mentions the regular involvement of the preauricular gland and the predominant appearance of the corneal changes in the pupillary area.

In some of the cases cited by him maculae corneae reduced the vision to 0.3 of the normal. Simultaneous occurrence of epidemic keratoconjunctivitis was reported, furthermore, by oculists in Augsburg and Erlangen in Bavaria, and in Düsseldorf in Prussia.

K. L. STOLL.

CHANGES IN REFRACTION OF THE CORNEA AFTER REMOVAL OF PTERYGIUM. S. DUDINOFF and L. TSEPENIUK, *Vestnik oftal.* 13: 100, 1938.

Dudinoff previously examined 500 patients with pterygia. He worked out a clinical classification and observed that a hypermetropic astigmatism develops because of flattening of the cornea in the horizontal meridian. In this study the authors show that the improvement of vision after the operation is due not only to the removal of the mechanical obstacle but to the change of refraction.

Fifty-two eyes were examined with the ophthalmometer before and after the operation. The astigmatism was lowered or disappeared completely after the operation in all cases. The authors believe that this occurred chiefly because of the increase of the refractive power of the cornea in the horizontal meridian and also because of the weakening of the refraction in the vertical meridian.

O. SITCHEVSKA.

Experimental Pathology

KERATOCONUS EXPERIMENTALLY PRODUCED IN THE RAT BY VITAMIN A DEFICIENCY. J. R. MUTCH and M. B. RICHARDS, *Brit. J. Ophth.* 23: 381 (June) 1939.

Thirty rats were used for the experimental production of keratoconus with a vitamin A free diet. Xerophthalmia developed in all in some degree. Because of complications, a number of the animals were useless for the purpose of the experiment. Keratoconus developed in 12 of the remainder. The ectasiae produced without exception were in the form of central cones. All the rats had acute xerophthalmia, but even in human beings this condition is noninflammatory. The majority had an anterior synechia, and it is thought that this might have been a factor in the production of the cones in those so affected. The authors do not suggest that keratoconus in the human being is due in every case to avitaminosis.

The authors submit the following summary:

"Keratoconus has been produced experimentally as a sequel to acute xerophthalmia in rats on a vitamin A-free diet. In most cases the cornea regained its normal contour after a few weeks dosing with vitamin A, corneal nebulae and myopia remaining as permanent defects."

The article is illustrated.

W. ZENTMAYER.

BEHAVIOR OF THE WATER CONTENT OF THE LENS IN EXPERIMENTAL CATARACTA PARATHYREOPRIVA. A. DE CRECCHIO, *Ann. di ottal. e clin. ocul.* 67: 59 (Jan.) 1939.

One eye of each of a series of 12 rabbits was enucleated before parathyroidectomy, and the other was enucleated at intervals of eight to forty-one days after this procedure. The second eye was observed with the slit lamp at weekly intervals before enucleation. The lenses were removed in the capsule, weighed, dried to constant weight and weighed again. In all except 1 animal, which was moribund on the eighth day, opacities developed, chiefly along the posterior suture lines. All showed an

increase in water content of the second eye, which averaged 14.30 per cent. With reference to the theory of Foerster, who grouped ions as positive and negative in a biologic sense as distinct from the physical sense, the author believes that a change in the equilibrium of the water-mineral exchange in the lens may produce a fall in the oxidation-reduction potential of the lens which would result in cataract.

S. R. GIFFORD.

CHANGES IN THE RETINA AFTER EXPERIMENTAL GASTRECTOMY ON DOGS. J. JENSEN, *Acta ophth.* 16: 649, 1938.

Fifteen dogs after resection of the stomach presented a peculiar clinical condition much resembling pellagra in human beings. They became anemic and showed loss of hair, pigmentation of the skin, disturbance of gait and stunted growth. Histologically, severe degeneration of the whole central nervous system was observed. The author examined the eyes of these animals and found degenerative changes of the third neuron of the optic pathway. The ganglion cells in the retina showed disturbed granulation and vacuole formation, while the optic nerves showed demyelination and glial proliferation.

The cause of these changes has not been elucidated but may be due to a lack of resorption of certain substances (probably vitamins) necessary to the nervous system.

O. P. PERKINS.

General Diseases

IRITIS AND OPTIC NEURITIS IN THE COURSE OF A SPIROCHETAL HEMORRHAGIC ICTERUS. J. BOLLACK and A. AURENCHE, *Bull. Soc. d'ophth. de Paris* 50: 580 (Dec.) 1938.

In the case reported the patient's initial symptoms were loss of vision with pain and redness of the eye. Both eyes on inspection revealed similar conditions. The pupils were irregular and adherent to the lens by synechiae. The anterior surface of the lens, viewed with the slit lamp, showed deposits of pigment. Cells were found in the anterior chamber. The ocular tension was normal. Floaters were in evidence in the vitreous. The papillae were hyperemic, with edematous borders. There was a concentric contraction of the visual fields. The visual acuity was 2/10 for each eye. Subsequently jaundice, anemia and itching developed. Examination of the serum was positive for syphilis. Under therapy, the ocular condition responded rapidly. The outstanding features of the case were the obvious diagnosis, the ocular complications and their rapid response to therapy.

L. L. MAYER.

Glaucoma

GLAUCOMA AND HYPERTENSION. A. MAGITOT, *Docum. ophth.* 1: 411, 1938.

The literature on the relation of hypertension to glaucoma is reviewed. The important distinction between glaucoma with hypertension and hypertension without glaucoma is carefully established. The effect of

hypertension on the ocular tissues, vision, light sense, the visual field, atrophy of the optic nerve and the fluid exchange in the eye is discussed. After scanning the literature, the author voices the opinion that the intraocular fluids are not derived from a single source but are produced by all the tissues of the eye and that glaucoma is an expression of an alteration in the permeability of the capillary system of the entire eye. The causal pathologic changes are therefore not limited to the region of the iridocorneal angle. Atrophy of the optic nerve is considered the result of an ischemia which produces a lacunar degeneration similar to the type seen in cerebral softening. Even normal tension may produce cupping in such a nerve head. The author concludes that the medical and surgical therapeutic measures which have withstood the test of time must be considered as acting not only on the hypertension but on the permeability of the capillaries, either directly or through the neural mechanism which controls the intraocular tension.

The bibliography is extensive.

E. G. SMITH.

IRIDECTOMY AB EXTERNO IN GLAUCOMA. Z. FRANK-KAMENETZKY.
Vestnik oftal. 13: 648, 1938.

The various forms of glaucoma and the proper operative procedures for each are discussed by Frank-Kamenetzky. He performs filtering operations on glaucomatous patients with deep anterior chambers and iridectomy ab externo on those with shallow anterior chambers (Roeder's classification of glaucoma). During the two years preceding this report iridectomy ab externo was done on 61 eyes after Elschmig's technic. The tension was increased in all cases, and it was reduced to normal after the operation in all but 1 case. The majority of the patients suffered from acute and chronic inflammatory glaucoma. The advantages of this operation are numerous: Since no instrument enters the anterior chamber (the iris as a rule prolapses in the wound and is cut off at the base), there is no danger of injuring the lens. The aqueous escapes slowly, so that there is no sudden change of intraocular pressure and spontaneous rupture of the capsule. The technic is simple and is described. Prolapse of the vitreous is a rare complication in this operation. It occurred only in 1 patient with a tension of 80 mg. of mercury.

O. SITCHEVSKA.

Injuries

TREATMENT OF LIME IN THE EYE. G. C. PETHER, Brit. M. J. 1: 668
(April 1) 1939.

The author emphasizes the importance of removing all large particles of lime from the eye as early as possible. This can be best done with a camel-hair brush, which is smeared with a mixture of equal parts of liquid petrolatum and petrolatum. The value of free and prolonged irrigation has always been recognized. A little liquid petrolatum is put in the eye, which is then covered with a pad.

In speaking of the nature of the injury caused by lime, the author states that the reaction of lime with water may cause a rise of temperature to 300 C., which suggests that the local production of heat is

responsible, at least in part, for the damage done by particles of lime in the eye. At the same time, with the use of a Gradenigo thermometer it has been shown that the temperature of the conjunctival sac has not been raised.

The author then discusses solvents which are at present in use and the solubility of lime in neutral salt solution. He is encouraged with the effect of the 4 per cent solution of ammonium chloride. This causes less irritation than the usual solution of boric acid. The addition of an analgesic to the irrigating fluid is helpful; in anesthetizing the eye a neutral anesthetic is less painful than cocaine hydrochloride or butyn, which are acid in solution.

The author comes to the following conclusions:

"The power of various neutral solutions to dissolve lime has been calculated by a series of experiments. It is shown that ammonium chloride is more effective than any solutions which have hitherto been generally employed. This solution in 4 per cent strength has been tried out on a series of cases with considerable success. It is no more painful to the eye than other irrigating fluids. The preliminary application of an analgesic solution and the removal of large particles by means of a camel-hair brush dipped in a mixture of vaselin and paraffin should also form part of the first-aid treatment."

ARNOLD KNAPP.

Lens

RADIATION CATARACT. A. TALIERCO, *Ann. di ottal. e clin. ocul.* 67: 104 (Feb.) 1939.

The literature concerning the pathogenesis of radiation cataract is reviewed. The author has attempted to learn whether chemical or physicochemical changes in the lens or aqueous humor are present which would throw light on the pathogenesis of this form of cataract. One eye of each of a series of rabbits was irradiated after atropinization, the rays falling perpendicularly to the plane of the iris through a lead plate which exposed only the cornea and the neighboring sclera. After two to five skin doses, either singly or in fractional exposures, all the animals showed opacities of the lens after from thirty-eight to one hundred and ten days, the opacities appearing sooner with the larger doses. The changes affected primarily the posterior cortex along the suture lines. An increase in weight of the irradiated lenses was observed varying from 4 to 8 per cent. There was a slight but constant shift of p_H in the aqueous toward the alkaline side and a slight but definite increase in the calcium of the lens. Analysis of the respiration of decapsulated lenses was carried out by Warburg's method. This showed a decrease in glycolysis in the irradiated lens but no change in oxygen consumption. All of these changes showed a direct relation to the degree of change in the lens. The change in p_H of the aqueous is attributed to the effect of roentgen rays on the vessels of the eye. The increase in calcium may result from an increase in permeability of the lens capsule. The lowering of glycolysis in the lens appeared only at the time when the opacities of the lens appeared and did not precede it. Hence it is probably a result of direct damage by the roentgen rays to lens epithelium, which generates new fibers, which effect is also, in all probability, responsible for the opacities of the lens.

S. R. GIFFORD.

BEHAVIOR OF SODIUM CHLORIDE IN THE AQUEOUS HUMOR AND IN THE BLOOD IN RESPECT TO PARATHYROIDECTOMY CATARACTS. F. NASTRI, *Rassegna ital. d'ottal.* 6: 19 (Jan.-Feb.) 1937.

The author studied the behavior of sodium chloride in the aqueous humor and in the blood of animals after parathyroidectomy in respect to the lenticular changes which are often seen in these cases. The results showed that the sodium chloride did not vary in the aqueous or in the blood before or after the operation.

A. PERZIA.

Neurology

THE TENTORIAL PRESSURE CONE. G. JEFFERSON, *Arch. Neurol. & Psychiat.* 40: 857 (Nov.) 1938.

This article should be interesting to ophthalmologists in that it points out how increased intracranial pressure can force the midbrain down into the hiatus tentorii against the free edges of the tentorium, producing signs referable to the midbrain, including fixed and anisocoric pupils, which are not referable to the site of the original lesion. In addition, many cases of hemianopia can be explained only by pressure on the optic tracts of an enlarged temporal lobe against the tentorium.

R. IRVINE.

CEREBRAL ANGIOMA ARTERIALE. H. H. HYLAND and R. P. DOUGLAS, *Arch. Neurol. & Psychiat.* 40: 1220 (Dec.) 1938.

In the case reported a vascular tumor encroached on the right optic radiation, producing a hemianopic quadrantic field defect. Periodic right-sided migrainous headaches had been present for many years. They were preceded by visual spots, and strong light or prolonged visual stimulation precipitated the attacks. Profuse lacrimation of the right eye frequently accompanied the headaches. An audible bruit increased after such stimulation, and the authors infer that use of the eyes increased the flow of blood in the tumor, leading to distortion and compression of the vessels, thereby causing the symptoms. In the general discussion of this type of tumor the reference to the occasional association of congenital abnormalities, such as buphthalmos, heterochromia iridis, coloboma of the optic nerve or high refractive error, should be interesting to the ophthalmologist.

R. IRVINE.

OCULAR DISTURBANCES IN LITTLE'S DISEASE. H. BÉGUÉ, M. BRISSOT and J. MAILLEFER, *Bull. Soc. d'opht. de Paris* 50:602 (Dec.) 1938.

Reports are found in the literature of involvement of the ocular musculature in from 30 to 44 per cent of cases of Little's disease. The authors report on the examination of 27 infants. Ten had normal eyes, 5 had myopia, 7 had nystagmus, 8 had convergent strabismus and 2 had divergent strabismus. Two patients had bilateral atrophy of the optic nerve. A discoloration of the papilla was found in 22 per cent. In agreement with other reports, 37 per cent had strabismus. Fournier considered the ocular defects as stigmas of hereditary syphilis. The

authors ascribe a more important role in the genesis of this syndrome to difficult labor followed by asphyxia, to tuberculosis, epilepsy, vesanic heredity by consanguinity and finally to diseases which develop during the first year of life.

A bibliography accompanies the article.

L. L. MAYER.

TRAUMATIC BITEMPORAL HEMIANOPIA (SAGITTAL TEARING OF THE OPTIC CHIASM). OSTERBERG, *Acta ophth.* 16: 466, 1938.

The author states that about 30 cases of bitemporal hemianopia following cranial injury have been reported in the literature. Gross rupture of the crossing fibers in the chiasm has been found in only 2 cases. Moreover, this type of lesion is not an incidental finding at the many postmortem examinations conducted on persons who have died as a result of injuries of this type. The author has tried the experiment of stretching a number of optic chiasms by means of two Prince forceps, varying the force and suddenness of the pull. Photomicrographs of the chiasms are shown; the important finding is multiple microscopic lacerations occurring in the middle part of certain of the specimens which show no macroscopic lesion. Osterberg believes this same type of lesion occurs when the anterior contents of the skull undergo sudden and violent lateral expansion.

O. P. PERKINS.

Operations

RESULTS OF RECONSTRUCTION OF THE EYELIDS AFTER REMOVAL OF TUMORS OR AFTER SEVERE TRAUMA. A. NICOLATO, *Ann. di ottal. e clin. ocul.* 67: 81 (Feb.) 1939.

Plastic surgical procedures for the eyelids are discussed in an article illustrated by forty-six photographs of patients. The author prefers to use sliding flaps whenever possible, since with flaps obtained by torsion or with pedunculated flaps the motility of the lids is usually unsatisfactory. Defects of one half or even two thirds of the lower lid can be repaired by sliding flaps, while for the upper lid such flaps are not satisfactory when more than one third of the lid must be sacrificed. When the tarsus is destroyed sliding flaps are not usually satisfactory, and pedunculated flaps must often be employed. Every effort is made to save the conjunctiva, as when it is preserved the tendency to shrinkage of the flaps is minimal. Blepharoplasty maintained for several months after operation is usually of advantage. By the use of double sliding flaps from above and below, according to the method of Imre and Blaskovics, large defects may be covered which would otherwise require pedunculated flaps. The method of Dieffenbach was employed in 15 cases, that of Knapp is 16 and that of Imre and Blaskovics in 9. A flap obtained by torsion was employed in 8 cases. In 3 cases the use of a tube flap from the neck, according to the method of Gillies, was necessary because of extensive destruction. In 1 case a free implant of skin and cartilage from the auricle was employed, with a good result. Definite indications for each of these methods exist in individual cases.

S. R. GIFFORD.

Orbit, Eyeball and Accessory Sinuses

BILATERAL ACCOMMODATION PARALYSIS AND UNILATERAL SCOTOMA IN SPHENOIDAL-SINUS DISEASE. N. B. ELLIS, *Am. J. Ophth.* 21: 1365 (Dec.) 1938.

Ellis reports the case of a 20 year old youth in whom bilateral paralysis of accommodation developed, followed by a unilateral retrobulbar neuritis, which cleared up miraculously after an operation on the sphenoid sinus. The literature is reviewed, and the following summary is given:

"1. This case presents the unusual combination of two ocular symptoms: right retrobulbar neuritis and bilateral accommodation paralysis, with X-ray diagnosis of right sphenoiditis in an anomalous sphenoid.

"2. Multiple sclerosis and encephalitis were ruled out as etiological factors.

"3. There was absence of rhinological evidence of sinus disease.

"4. The importance of free opening and exploration of the sinuses in visual disturbance is stressed."

W. S. REESE.

Physiology

DETERMINATION OF THE THRESHOLD SENSIBILITY OF THE RETINA. A. CHEVALLIER and H. ROUX, *Compt. rend. Soc. de biol.* 130: 1279, 1939.

In the adaptometer previously described by the authors the illumination consists of a definite hue of spectral blue. After thirty minutes of dark adaptation, the diaphragm is widened until the subject just perceives the sensation of light, and the diameter of the diaphragm at this point is taken as the index of the person's light threshold. The results are constant. One normal person measured eleven times in different days maintained a threshold of 3.8 mm. and another, 6.3 mm. The measurement in a case of night blindness was constant at 52 mm.

J. E. LEBENSOHN.

INFLUENCE OF DARK ADAPTATION ON THE CRITICAL FREQUENCY OF FLICKERING OF MONOCHROMATIC LIGHT. S. KRAVKOV, *Vestnik oftal.* 13: 72, 1938.

The purpose of the experiment reported here was to establish the change of the critical frequency of flickering in the course of dark adaptation for monochromatic irritants of various wavelengths. The method of the experiment is described, and several drawings and diagrams illustrate the article, which lends itself to abstracting with difficulty.

The following conclusions are drawn:

1. The frequency of flickering is decreased during the course of dark adaptation.

2. The initial critical frequency at the beginning of dark adaptation is decreased chiefly by blue irritants and less by orange and red, and it hardly changes on stimulation with green.

3. Comparison of the curve of the relative lowering of the critical frequency of flickering for various monochromatic irritants with curves of the three basic irritants of the eye shows that the blue-sensitive apparatus of the eye gives the highest decrease of the critical frequency, while little change is produced by its green-sensitive apparatus.

4. The experiment on protanopes shows a considerable lowering of the critical frequency by blue irritants, while it remains on the same level for all other rays of the spectrum; this fact indicates that the green-sensitive and red-sensitive apparatus of the protanope's eye is not differentiated.

5. It is possible that at the basis of the lowering of the critical frequency of flickering lies the lowering of the differential sensitivity of the eye which is connected with the nerve centers above.

O. SITCHEVSKA.

Retina and Optic Nerve

THE RETINAL CIRCULATION. P. BAILLIART, *Docum. ophth.* 1:161, 1938.

A comprehensive review of the literature on the functional aspects of the retinal circulation is undertaken. The first part of the article deals with anatomic considerations, nerve control, effects of drugs, measurements of pressure, pressure equilibrium and modifying factors as related to the retinal circulation. The second part deals with the effects of pathologic changes on the retinal circulation. The mechanism and causation of edema and hemorrhages in various diseases and intoxications are outlined. There is an extensive bibliography. The pathologic changes are not touched on.

E. G. SMITH.

MOBILE EMBOLUS BETWEEN TWO BIFURCATIONS OF THE ARTERIES. BAILLIART, KALT and O. DE SAINT-MARTIN, *Bull. Soc. d'opht. de Paris* 50: 576 (Dec.) 1938.

A man aged 66 had sudden and complete blindness of the right eye. General physical examination revealed only headache, dizziness, polyuria and moderate hypertension. The fundus of the right eye gave the classic picture of thrombosis of the central vein. The left eye showed marked arteriosclerosis. The superior temporal artery above its first bifurcation was dilated, resembling a small aneurysm. When pressure was applied to the globe in taking a dynamometer reading the dilatation was obliterated, the artery to the next bifurcation paled and on release of pressure the original condition returned. By regulating the pressure it was demonstrated that a small embolus migrated from the dilated first bifurcation down to the second bifurcation and subsequently returned to its former position.

L. L. MAYER.

JUXTAPAPILLARY RETINOCHOROIDITIS WITH NARROWING OF THE VISUAL FIELD IN THE SUPERIOR SECTOR. J. BOLLACK and A. WIOZ, *Bull. Soc. d'opht. de Paris* 50: 586 (Dec.) 1938.

It is not unusual in Jensen's disease to locate a scotoma in direct relation to the lesion near the nerve head. The authors found a loss in

almost the entire superior half, except for a small temporal sector. A photograph of the lesion in the fundus and the visual field loss is included in the article.

The patient, a woman of 27 years, had noted diminution of vision for one year. The lesion, which was found in the left eye only, was typical except for the fact that it extended somewhat below the disk. The visual acuity was 9/10. The causation was not determined. The authors are inclined to believe that in spite of so little involvement and so little reaction, the fibers approaching the disk from below had been degenerated by the process.

L. L. MAYER.

TREATMENT OF OBSTRUCTION OF THE RETINAL VESSELS BY THE RETROBULBAR INJECTION OF ACETYLCHOLINE FOLLOWED BY INTRAMUSCULAR INJECTION OF COBRA VENOM. MICHAUX, Bull. Soc. d'opht. de Paris 50: 605 (Dec.) 1938.

Interest in the choline derivatives has increased in the past few years. The indication has been to accomplish vasodilatation in such conditions as obstruction of the vessels, intoxications, certain types of retinal hemorrhages, glaucoma, circinate retinitis and ophthalmic migraine. The usual method has been the subcutaneous injection of acetylcholine. Michaux has obtained more rapid and constant results with massive doses of acetylcholine injected retrobulbarly. At the first injection 5 cg. is used unless the condition is of long standing, and then 10 cg. is used. If the first injection causes no relief, it may be repeated as often as three times during the week. In the successful cases recovery occurs within a few minutes after the injection. As the results have been variable, a drug was sought to reenforce the effects of the acetylcholine, and cobra venom was chosen. It is well known that an intramuscular injection of 0.1 mg. of cobra venom lowers the pressure of the cerebrospinal fluid and produces a marked and durable elevation in the retinal arterial pressure.

Complications due to retrobulbar injection of acetylcholine may be numerous. Edema of the conjunctiva and lids, Horner's syndrome, paralysis of the extraocular muscles and stimulation of the oculocardiac reflex with syncope, bradycardia, nausea and vomiting may occur. Seven case reports are given in detail. Michaux concludes:

1. To obtain a rapid amelioration of obstruction of a retinal vessel a retrobulbar injection of from 5 to 10 cg. of acetylcholine is indicated.
2. Cobra venom augments the action of the acetylcholine.
3. The finding of a Horner's syndrome after injection is favorably prognostic in that one may be sure the drug has acted on the terminals of the cervical portion of the sympathetic nervous system.
4. The more recent the lesion, the more favorable the prognosis.
5. Of the 7 patients treated by the combined method, 2 showed complete cure and 3 partial cure; vision of only 1/10 was recovered by the other 2 patients.

L. L. MAYER.

Trachoma

STUDIES ON TRACHOMA. L. DE ANDRADE, *Ann. d'ocul.* 176: 33 (Jan.) 1939.

In 1935 von Szily, at the University of Münster, discovered the possibility of making cultures of trachoma in the eyes of laboratory animals by injecting into the vitreous a few drops of human trachomatous material. Rabbits, chickens and guinea pigs were used. Von Szily observed the formation of follicles in the choroid, resembling histologically the follicles of trachoma. These were also found in the brain of some of the animals.

The importance of this discovery was not seriously noticed by the trachoma workers. After von Szily, Busacca made similar reports. In 1935 the author published an article showing that injection into the vitreous of substances with a chemical constituent, such as cholesterol, lecithin and phytine, produced in the center of the uvea lymphatic follicles, sometimes with giant cells. He believed this histologic reaction depended on the molecular structure of the substance employed.

In some work that the author instituted, with a view to verifying the possibility of carrying human trachoma into the eye of the rabbit, he used fresh material from trachomatous granulomas and mixed this immediately with some drops of physiologic serum. He injected this mixture in amounts of 0.1 to 0.2 cc. The inoculations were positive in that microscopic examination showed lymphocytic nodules in the choroid, while the appearance of the fibrous tissue demonstrated an essential trachomatous reaction.

Trachoma as a general malady is then described, with the writer's personal investigations, and the results of examination of the blood are presented in two tables. He concludes that it is possible to carry human trachoma into the eye of the rabbit. The blood picture of trachoma always reveals an important lymphocytosis, and the alkaline and the calcium reserve of trachomatous patients are normal.

S. H. McKEE.

ORIGIN OF THE FOLLICULOMA AND THE FOLLICLES IN THE DENIG TRANSPLANT: REPORT OF CASES. C. PASCHEFF, *Klin. Monatsbl. f. Augenh.* 101: 361 (Sept.) 1938.

Pascheff refers to his previous research which proved that trachoma is not an exudative process but a hyperplasia of a follicular and confluent nature. Later he proved the occurrence of these hyperplastic formations on the cornea, which he called folliculomas. This new term replaced the terms of granulations or granulomas, with reference to the conjunctiva, and pannus crassus, or pannus sarcomatosus of the cornea. Pascheff reported a number of cases, 1 of them being a case of beginning folliculoma in which there were some distinct germinal centers of follicles. These centers become confluent and degenerate at a later stage of trachoma. Hence the author deducts that the folliculoma consists of a continued formation and steady growth and confluence of follicles. In some cases Pascheff observed that folliculomas recurred in buccal mucosa transplanted into the conjunctiva after Denig's method. This phenomenon cannot be explained as a result of the contagiousness

of trachoma. It originates, in Pascheff's opinion, through the intervention of blood vessels, the formation of which precedes the formation of follicles. Follicles form around the blood vessels and where the most blood vessels are present, for example, in the fornix. In this connection the absence of adenoid tissue in the cornea is of no consequence, but the mere presence of blood vessels is sufficient.

Trachoma is of a hyperplastic nature. It cannot develop without follicles, and the trachomatous follicle is a germinative, confluent follicle. It remains uncertain whether the soil or a virus is responsible for the formation of folliculomas, the final products of this evolution.

K. L. STOLL.

SURGICAL TREATMENT OF TRACHOMATOUS PANNUS OF THE CORNEA.

E. MORETTI, *Klin. Monatsbl. f. Augenh.* 101:373 (Sept.) 1938.

Moretti discusses the large number of surgical methods devised to combat trachomatous pannus of the cornea. Their results have not been satisfactory. He originated a new operation, called perirrhaphy, which he employed in 83 cases, 70 of which were cases of thick pannus of the cornea and 13 of which were cases of what he calls dry pannus. The thick pannus disappeared after twenty to forty days, according to the thickness, in all but 6 cases. Vision increased to normal in some of the author's cases. The method consists in the insertion of a black silk thread about 3 mm. from the limbus which is carried around the cornea circularly. Thus a ring is formed in the episcleral tissue by the thread, the ends of which are cut 5 to 6 mm. from the knot, which is tied toward the temporal canthus. The favorable result of this operation is attributed to the hyperemia produced by the thread, which is left in position for from twenty to thirty days. This method may be employed safely in the presence of corneal ulcers, which present too much risk for the use of other methods.

K. L. STOLL.

Tumors

TUMOR OF THE REGION OF THE INFERIOR CANALICULUS. P. DESVIGNES and P. BRIGÉAT, *Bull. Soc. d'opht. de Paris* 50:590 (Dec.) 1938.

The patient had an excrescence everting the orifice of the inferior canaliculus. The clinical aspect was one of an intracanalicular adenoma, which in growing had evaginated the canaliculus and everted its orifice. Because of its rarity, the suspicion that it might be malignant was cause for a biopsy. Histologic examination presented a diagnosis of tuberous nevus. The growth was removed in toto by diathermy.

L. L. MAYER.

PRIMARY SEBACEOUS EPITHELIOMA OF THE MEIBOMIAN GLANDS.

R. PAGÈS, E. STORA and J. DUGUET, *Bull. d'opht. de Paris* 50:621 (Dec.) 1938.

In 1936 Morard presented before the Ophthalmological Society of Paris his remarkable study of sebaceous tumors of the lids with a useful

anatomicopathologic classification. The condition in the case presented here is of interest because of its lobulated nature and its origin from the acini of the glands. This tumor was noted as an increasing lump in the lower lid of a man 33 years of age. There was no pain or loss of function. The only complication was the enlargement of the preauricular gland to the size of a pea. Photographs of the patient and the tumor are included in the article. After dissection, the growth recurred and disappeared finally after radiation therapy. Many mitotic figures were found in the cells partaking of the acini of the meibomiam glands. The preauricular gland was excised, and metastatic cells could be demonstrated. Clinically, such a tumor is most usually found on the upper lid. The malignancy of the tumor was unexpected.

L. L. MAYER.

MELANOSARCOMAS OF THE IRIS. P. SIEGERT, Arch. f. Ophth. 139: 591 (Dec.) 1938.

The author reports the clinical and pathologic observations made on 3 cases of sarcomatosis of the iris without involvement of the rest of the uvea. In the first case a small sarcoma which had arisen from a nevus was discovered accidentally in the course of a routine pathologic examination. In the other 2 cases the sarcomatosis was diagnosed in vivo and pathologically was found to have its substrate in malignant pigmented tumors which grew chiefly in the plane of the iris. The observations made on these 3 cases confirm the classic concept of the origin of sarcomas from benign melanomas. This mode of development is not always recognizable by clinical observation. One should distinguish between anatomic malignancy (infiltration, destructive growth and metastases) and clinical malignancy characterized by the development of secondary glaucoma and loss of visual function. A benign tumor of the iris in the pathologic sense may be very malignant from the point of view of the clinician. The growth of the tumor may be so diffuse that the clinical symptoms are those of secondary glaucoma, with perhaps slight heterochromia or irregularity of the pigmentation of the affected iris. In such cases one should always bear the possibility of tumor in mind and in case of doubt perform a diagnostic iridectomy. Sarcoma of the iris seems to react temporarily but not permanently to irradiation (roentgen rays). The tumor grows slowly and metastasizes through lymph channels. Spreading of the tumor through the channels of outflow of the aqueous seems to be inhibited by some physicochemical changes which the tumor cells undergo in the aqueous. It is often difficult to determine with certainty from which normal tissue element of the iris such tumors are derived, but an origin from mesodermal elements is most probable. In addition to the small definitely malignant cells which make up the bulk of the tumor, one often finds a proliferation, not definitely malignant, of large melanophores the origin and significance of which are uncertain.

The paper is well illustrated with photographs of the microscopic slides and of the picture in vivo.

P. C. KRONFELD.

Uvea

TREATMENT OF GLAUCOMATOUS STATES IN ANIRIDIA. JEAN-SEDAN, Bull. Soc. d'opht. de Paris 50: 615 (Dec.) 1938.

Fifteen patients with aniridia have been observed in consultation during the past twenty years. Of these, 3 have had glaucoma. The association of glaucoma with aniridia is a fairly rare condition, and little reference has been made to it in the literature. Vannas in 1932, Scalinci in 1904 and Dennis in 1906 have reported on experimentally produced aniridia and the effectiveness of mydriatic and miotic drugs. They all concluded that miotics reduce the tension to a greater degree in eyes with aniridia than in normal eyes. Reports of 2 cases are given in detail. Jean-Sedan advises a filtration operation with partial iridectomy in the region where a portion of the iris remains.

A bibliography accompanies the article.

L. L. MAYER.

Therapeutics

CONCENTRATED, FRACTIONAL ROENTGENOTHERAPY AT CLOSE RANGE IN MALIGNANT TUMORS OF THE EYELIDS. R. BRAUN, Klin. Monatsbl. f. Augenh. 101: 557 (Oct.) 1938.

A synopsis on irradiation at close range after Chaoul's method and on its technic is given, including references to the research of other authors. Braun describes his own technic, with which he treated 11 patients suffering from carcinomas of the eyelids. All but 3 patients recovered. Adverse symptoms, observed by other writers, were not met with; a lead prothesis was applied for the protection of the healthy tissues. Surgical intervention may be avoided in most cases by the use of Chaoul's method, which may replace the treatment with thorium X rods.

K. L. STOLL.

TREATMENT OF THROMBOSIS IN CENTRAL VEIN OF RETINA WITH HEPARIN. K. PLOMAN, Acta ophth. 16: 502, 1938.

The author uses a 5 per cent solution of heparin intravenously. From 100 to 150 mg. of the drug are administered twice daily for a period of eight or ten days. He reports his results in 2 cases of thrombosis of the trunk of the central retinal vein and in 6 cases of thrombosis of its branches.

Recovery occurred in 1 case of thrombosis of the trunk in the short period of twelve days. Objective, rapid improvement also occurred in the other case.

In 1 case of thrombosis of a branch there was no improvement. In the remaining 5 cases there was an average improvement in vision of 3/10 in from one to fourteen days.

The author speculates as to how heparin exerts its favorable action. It is known to retard coagulation and so may prevent the addition of new fibrin to a thrombus, thus preventing a partial thrombus from becoming complete. Moreover, if the efferent flow of blood is facilitated, vitality of tissue is improved and possibilities of resorption are increased. Heparin probably passes through capillary walls into tissues and may contribute toward keeping newly extravasated blood more fluid and more easily absorbable.

O. P. PERKINS.

Society Transactions

EDITED BY W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY

DAVID WEBSTER, M.D., *Chairman*

Oct. 16, 1939

ROBERT K. LAMBERT, M.D., *Secretary*

SARCOID OF THE LACRIMAL GLAND. DR. DAVID WEXLER.

A Negro, aged 29, was treated five years prior to this presentation for early pulmonary tuberculosis. In the last three years both eyes became prominent, particularly the left. In each lacrimal area there was a firm mass. A portion of the mass in the left orbit was removed. Microscopic section revealed so-called hard tubercles, without caseation and considerable calcium deposit. Only a few lacrimal tubules remained. Sections stained for tubercle bacilli failed to disclose the organism. The lesion was considered to be identical with that usually described in cases of sarcoid.

DISCUSSION

DR. TOWNLEY PATON: I wonder what the impression is about the use of tuberculin in the treatment of sarcoid. An article on this question appeared three or four years ago in the *American Journal of Ophthalmology*. As a pathologic report was given, I do not believe that there is any question but that the growth in the case reported was a true sarcoid. The nodules disappeared after the use of tuberculin. They were in the subcutaneous tissue and not in the periosteum.

DR. DAVID WEXLER: The general opinion is that patients suffering from sarcoid do not give a positive reaction to a tuberculin test and do not respond to treatments with tuberculin. In fact, this is said to be one of the distinguishing points between true tuberculosis and so-called paratuberculous lesions. Perhaps the response in the case mentioned by Dr. Paton was due to a nonspecific effect of the foreign protein.

STREPTOTHRIX INFECTION OF THE LOWER CANALICULUS: REPORT OF A CASE. DR. DAVID O. SHEPARD.

HYPERTROPHY OF THYMUS IN VERNAL CONJUNCTIVITIS. DR. GUSTAVO ALAMILLA, Habana, Cuba.

This paper will be published in full, with discussion, in a later issue of the ARCHIVES.

CHORIORETINAL ARTERIOLAR NECROSIS IN MALIGNANT HYPERTENSION: REPORT OF A CASE. DR. MARTIN COHEN.

This paper will be published in full, with discussion, in a later issue of the ARCHIVES.

Book Reviews

Investigations sur le glaucome (Essais). By E. P. Fortin. Pp. 47, with 34 photomicrographs. Buenos Aires, "El Ateneo," 1939.

This monograph of Fortin summarizes in a conclusive and interesting way articles by him on the subject of glaucoma which have been published in periodicals not easily accessible to ophthalmologists. The book, consisting of 47 pages, with many illustrations, gives a description of the anatomic and physiologic changes in the anterior part of the eye in relation to glaucoma. The vast amount of literature connected with this problem cannot be considered in so short an essay. The book gives only the results of Fortin's exact anatomic studies and his explanations, which are sometimes widely divergent from those which science has accepted as proved.

The statements presented in this paragraph have been taken from Fortin's monograph: The comparatively strongly developed ciliary muscle lying between the sclera and the epithelium moves the "irido-ciliary" fluid into the cavities of the iris and into the ciliary processes. The division of the muscle fibers into longitudinal and radial types is a mistake, caused by artefacts. Between the two types, intermediate fibers are found, proving that the respective groups belong together. The muscle fibers which extend farthest back are connected with the walls of the large choroidal veins by tendinous strands. Other anatomic relations between the ciliary muscle and the vessels are also of great importance. The long posterior ciliary arteries traverse the ciliary muscle in its entire length before dividing in two branches, which form the *circulus arteriosus*. Contraction of the muscle may therefore throttle the supply of arterial blood. On the other hand, since the veins pass on the surface, its contraction will dilate them. The same effect is to be expected in the veins of the choroid on account of the anatomic relations mentioned.

Fortin describes in detail the anatomic structure of the *ligamentum pectinatum*, which he prefers to call *filtre pectiné*, and of the canal of Schlemm. The changes in shape of Schlemm's canal and the action during contraction and relaxation of the muscle are illustrated by excellent pictures of cross sections. During the contraction of the muscle, an expansion of the filter is accompanied by a tube-shaped dilatation of Schlemm's canal.

The mechanical action of miotics is clearly understood in the light to these anatomic and physiologic statements. The threefold action of the ciliary muscle—throttling of the arterial circulation, dilatation of the veins and the unfolding of the *filtre pectiné*—can also be caused by miotics. Each contraction of the ciliary muscle, which Fortin compares with the heart muscle, moves fluid from the anterior chamber into the canal of Schlemm. In acute attacks of glaucoma iridectomy should

facilitate the escape of iridociliary fluid which circulates in the cavities of the iris and the ciliary body and which communicates with the spaces of Fontana. However, a normal *filtre pectiné* is a condition sine qua non.

L. SALLMANN.

Atlas clinique d'ophtalmoscopie photographique. By Henri Tillé, M.D., and A. Couadau, M.D. Price, 280 francs. Pp. 194, with 202 illustrations. Paris: Masson & Cie, 1939.

As photography of the eyeground has made great advances, the authors believe that an atlas of photographic pictures of the fundus oculi would be suitable for young ophthalmologists and general practitioners to learn that the elementary lesions in pathologic conditions of the fundus are comparatively simple and conform to similar changes encountered in general pathologic conditions. The apparatus used is the well known Nordenson camera. The great advantage of photography of the fundus, as has been stated before, is that successive pictures will describe the course of a lesion and at the same time give exact measurements of the lesion, while the drawings in color of the older atlases lack the exactness of a good photograph.

The photographs were obtained during ten years' attendance at the clinic of Dr. Bailliart at the Quinze-Vingt, and a number of others have been added through the courtesy of Amsler, Bailliart, Diaz-Caneja, Laignier, Mawas, Pavia and Schiff-Wertheimer. The authors are of the opinion that color photography today does not seem sufficiently advanced to be of use in such an atlas.

The atlas is divided into the following sections: normal eyegrounds; traumatic lesions of the chorioretina and optic nerve; inflammatory lesions of the optic nerve and of the choroid; aplasia and coloboma of the fundus; degenerative lesions of the retina at the macula; myopic chorioretinitis; retinal detachment; hypertensive and azotemic retinopathies; diabetic and associated retinopathies; islands of disintegration, softening of the retina and sclerosis of the choroidal ring; angioid streaks of the choroid (Groenblad's disease); leukemic and erythematous retinitis; cyanotic retina in Roger's disease; changes in the retina and nerve head in increased intracranial and intraocular pressure, and tumors of the optic nerve, retina and choroid.

Each chapter has a brief introduction, giving the main changes in the fundus picture, their histologic appearance and their clinical course. Each photograph is accompanied by a brief clinical case history and a description of the changes in the fundus. Thus the plan of the atlas is comprehensive, instructive and excellent, and the photographs reproduced in black and white are as good as this mode of illustration permits. The 202 photographic illustrations are not all equally effective, and some may offer difficulties of interpretation for the beginner.

The authors are to be complimented on the publication of a most useful atlas, and the publishers, for beautiful book making.

ARNOLD KNAPP.

Rétine humaine et phénomènes entoptiques. By E. P. Fortin. Price, paper, 250 francs. Pp. 196, with 124 photomicrographs. Buenos Aires: Vigot Frères, 1938.

Fortin's monograph, published in a limited edition, presents the results of extremely accurate research work which he has carried on for almost thirty years and published in a great number of articles in different periodicals.

The first and most important part deals with so many problems of the retina that only a brief review of the outstanding findings can be given—a review which shows clearly the revolutionary character of Fortin's statements and ideas.

Fortin found that the pigment epithelium of the retina appears on cross section in the form of small disks. The cells are not hexagonal. Rods and cones are equal in length. The neuroepithelium of the macular region is distinguished by its uniformity. Its elements should not be defined as cones but as a special structure with intermediate characteristics. Fortin considers the *membrana limitans externa* as a fine uninterrupted membrane with minute openings which is connected with the ciliary muscle at the *ora serrata*. In this way changes in the form of the macula may be caused by the action of the ciliary muscle.

Great emphasis is laid by Fortin on the anatomic structure and function of Henle's fiber layer, underestimated by Cajal and his pupils. The fine fibers of this layer are probably thin tubes, which are suspended in the intraretinal fluid and serve in the transportation of this fluid. Peculiar formations called *campanules*, bell-shaped cells, are in close contact with Henle's fibers. The *campanules* lie in the layer of small structures; these are uniform-sized elements which react to light stimuli. The Henle fibers and the small structures are carriers of light sensation. Fortin does not consider the neuroepithelium a receptive layer. Müller's fibers, according to Fortin, are misinterpreted in the usual histologic studies. He denies the existence of these structures and calls them artefacts.

An especially detailed description is given of the fine capillaries with a uniform caliber of 4 microns lying in the inner granular layer. Red cells, therefore, can pass through these walls only if flattened. The macula does not have these capillaries. The intergranular layer contains the aforementioned intraretinal fluid in which the fibers of this layer are immersed, but there are no cells.

Fortin emphasizes the primary importance of the anatomic structure of the macula for the interpretation of the human retina. To study the points in question he stresses the need of careful and improved technic applied to fresh normal human eyes.

The second part of the book treats of entoptic phenomena. A short historic introduction leads to a general review of the method of entoptic tests. A detailed description is given of different entoscopes invented and applied for various purposes by the author. Among the entoptic phenomena produced by various structures of the eye, the cornea, lens,

vitreous and retina, those of the retina are of special interest. The author tries to apply them to prove his interpretation of the anatomic changes which are described in the first part of the book.

L. SALLMANN.

Surgery of the Eye. By Meyer Wiener, M.D., and Bennett Y. Alvis, M.D.. Price, \$8.50. Pp. 445, with 396 illustrations. Philadelphia: W. B. Saunders Company, 1939.

The book is dedicated to Dr. Charles E. Michel, whose picture appears as a frontispiece, entitled "A Master Eye Surgeon." The authors have aimed to write a short and practical guide on operations on the eyes for the practicing ophthalmologist and student of ophthalmology; they definitely state that the book is not to be exhaustive but only to contain the methods which in the authors' judgment and experience have served to the best purpose. This immediately focuses the reader's interest and attention and gives the book a personal appeal. Moreover, to correct the error that books on operative surgery are generally insufficiently illustrated, the feature of diagrams is stressed, and 396 illustrations are introduced as a result.

The reviewer would like to draw attention to the following features: In the beginning a method of threading and transfixing needles is fully described. In speaking of the advantages of operating in the afternoon, the authors make no mention of the fact that the principal advantage is that the patient is nearer ready for the usual night's sleep, as pointed out by H. Knapp. The Smith method of intracapsular extraction, on page 84, is not correctly given and the diagram, figure 59, does not show the tumbling procedure. Though operations for ripening immature cataract are generally no longer popular, the authors prefer the direct massage (Bettmann) operation. The advantages of the Ziegler through and through operation for congenital cataract are emphasized. Herbert's iris inclusion operation is fully described and recommended for cases of glaucoma when other operations have failed. Operations for detachment are not fully enough described. The present plan of operators with the greatest experience in operations for detachment is to limit the coagulation to the pathologic area in the retina, and extensive delimiting procedures are no longer practiced. To leave the eye open and have the patient wear Lindner's stenopaic spectacles after twenty-four to forty-eight hours seems hardly wise.

Intraocular foreign bodies are all extracted by the posterior route, though no mention is made of the subsequent detachment which so frequently occurs.

The authors' method of resection or peeling off of the opaque portion of the cornea for corneal scars is fully described, and it is said to give as good if not better results than the now so popular corneal transplantation.

The authors' method of peripheral resection of the cornea for keratoconus is described. A method of late implantation after enucleation is interesting.

The authors like to utilize strips of fascia in operations for ptosis.

The Wiener-Sauer technic of nasal dacryocystorhinostomy is then described.

The names of authors are frequently given in the text but it would be an advantage if the exact literary references were added.

The authors have written a readable and instructive textbook. The matter is presented in a clear, concise way, and, what is important, the authors' point of view is always made clear. While some of the methods described, particularly those which the authors have devised or modified, may seem rather complicated for the average operator to follow, they will be instructive to the experienced surgeon. The general principles are sound, and the tendency to simplify the operative technic should particularly appeal to beginners. The illustrations are excellently chosen and add greatly to the book's usefulness.

ARNOLD KNAPP.

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Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. G. Woodward, 100 S. James St., Goldsboro.

Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.

Secretary-Treasurer: Dr. F. L. Wicks, 516-6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. O. Clement, 406 State St., Salem.

Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.

Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
 Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
 Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m.,
 second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
 Secretary: Dr. J. W. Jervy Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
 Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg.,
 Memphis.

TEXAS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. A. N. Champion, 705 E. Houston St., San Antonio.
 Secretary: Dr. Dan Brannin, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. A. E. Callaghan, Boston Bldg., Salt Lake City.
 Secretary-Treasurer: Dr. Rowland H. Merrill, 1010 First National Bank Bldg.,
 Salt Lake City.
 Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of
 each month.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
 Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd. S. W., Roanoke.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE
 AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
 Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
 Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
 Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Arthur G. Fort, 478 Peachtree St. N. E., Atlanta, Ga.
 Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
 Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month
 from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital,
 Baltimore.
 Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
 Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m.,
 fourth Thursday of each month from October to May.

BIRMINGHAM EYE, EAR, NOSE AND THROAT CLUB

President: Each member, in alphabetical order.

Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.

Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.

Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.

Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.

Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.

Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.

Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.

Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.

Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.

Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI OPHTHALMIC CLUB

Chairman: Each member, in rotation.

Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.

Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

CLEVELAND OPHTHALMOLOGIC CLUB

Chairman: Dr. Albert J. Ruedemann, Cleveland Clinic, Cleveland.

Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.

Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.

Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.

Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Harry M. Sage, 9 Buttles Ave., Columbus, Ohio.

Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.

Place: The Neil House. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.

Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.

Time: Second Thursday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Maxwell Thomas, Medical Arts Bldg., Dallas, Texas.
 Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
 Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
 Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
 Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
 Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
 Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
 Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
 Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
 Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
 Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
 Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
 Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
 Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
 Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
 Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
 Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.
 Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
 Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
 Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
 Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
 Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
 Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
 Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
 Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St. N. W., Washington.
 Secretary: Dr. Elmer Shepherd, 1606-20th St. N. W., Washington.
 Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
 Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
 Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Herbert G. Smith, 411 E. Mason St., Milwaukee.
 Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
 Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
 Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
 Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
 Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
 Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
 Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St. W., Montreal, Canada
 Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, 119-7th Ave., Nashville, Tenn.
 Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
 Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW HAVEN OPHTHALMOLOGICAL SOCIETY

President: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
 Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
 Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
 Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. James W. White, 15 Park Ave., New York.
 Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
 Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Arthur M. Yudkin, 257 Church St., New Haven, Conn.
 Secretary: Dr. Benjamin Esterman, 515 Park Ave., New York.
 Place: Squibb Hall, 745-5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND
 OTO-LARYNGOLOGICAL SOCIETY

President: Dr. J. T. Maxwell, 1140 Medical Arts Bldg., Omaha.
 Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
 Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
 Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
 Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
 Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
 Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Adolph Krebs, 509 Liberty Ave., Pittsburgh.
 Secretary: Dr. George H. Shuman, 351-5th Ave., Pittsburgh.
 Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
 Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
 Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
 Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
 Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second
 Wednesday of each month from September to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. B. Y. Alvis, Carleton Bldg., St. Louis.
 Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
 Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and
 scientific meeting 6:30 p. m., fourth Friday of each month from October to
 April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
 Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio,
 Texas.
 Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month
 from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE,
EAR, NOSE AND THROAT

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
 Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
 Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tues-
 day of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. I. Henry Smith, Slattery Bldg., Shreveport, La.
 Secretary-Treasurer: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
 Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every
 month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter W. Henderson, 407 Riverside Ave., Spokane, Wash.
 Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
 Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of
 each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
 Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
 Place: University Club. Time: First Tuesday of each month except June, July
 and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
 Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
 Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month,
 November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. Ernest Sheppard, 927 Farragut Sq. N. W., Washington, D. C.
 Secretary-Treasurer: Dr. E. Leonard Goodman, 1801 I St. N. W., Washington,
 D. C.
 Place: Episcopal Eye and Ear Hospital. Time: 7:30 p. m., first Monday in
 November, January, March and April.

ANALYSIS OF OPERATIVE RESULTS IN CONCOMITANT CONVERGENT STRABISMUS

GLEN GREGORY GIBSON, M.D.

PHILADELPHIA

There are two particularly important decisions which have to be made in regard to operations for concomitant convergent strabismus. The first and most important of these is the selection of the proper muscles for the operation, and the second is the determination of the amount of operative correction which should be done. In an attempt to improve the results of such operations two series of patients were operated on by members of the ophthalmic staff. The patients comprising the first series were subjected to bilateral recession of the internal rectus muscles. For those patients in the second series unilateral recession of the internal rectus muscle was combined with resection of the external rectus muscle of the same eye. On completion of these series of operations, analysis of the preoperative, operative and postoperative data was undertaken. From this material some general principles have been derived which may be of assistance in the solution of future problems. When operative measures are indicated, it is necessary to select the proper type of procedure to meet the demands of the muscular abnormalities in the particular case. This selection implies the proper use of those surgical procedures which diminish and those which increase muscular action. It is also necessary to apply these two types of procedures to the four main horizontally acting muscles. It follows that the correct application of the two main types of surgical procedures to the four main horizontally active muscles requires a knowledge of the functional status of the muscles in the particular case. This is necessary so that muscles which are functioning excessively may be subjected to a procedure that will diminish their action and so that those muscles the action of which is insufficient may be subjected to the procedures that will improve their action. Furthermore, it is necessary that these various procedures be properly graduated in amount in each individual case.

From the Department of Ophthalmology, Temple University School of Medicine.
Read before the College of Physicians of Philadelphia, April 20, 1939.

A discussion of this article appeared in the transactions of the society, in the September issue of the ARCHIVES (22:511, 1939).

The material for this study was composed of 66 patients, of whom 44 were operated on by the bilateral recession technic and 22 were operated on by the recession-resection procedure. The youngest patients (4) in this group were 4 years of age, and one fourth of the entire group were under 8 years of age. The rest of the patients were between 8 and 25 years of age. Since the deviation was present for two years or more in all these cases at the time of the first examination, the various sequelae of strabismus were well established. In view of this, a complete cure of the sequelae was neither attempted nor achieved. For these reasons the main surgical objective was a cosmetic improvement rather than the more ideal establishment of binocular single vision. The operative procedures in each series were done on consecutive patients, and selection of the procedure was not done as a rule. This plan of procedure was carried out for comparative purposes.

The purpose of this report is to give the results of the analysis of this group of operations. The operative treatment for early strabismus is not included in this report. No attempt is made to report new or modified procedures or to discuss the relative merits of various types of operations on the muscles. The literature is so extensive on this subject that it has not been included, but it is well discussed in such recent publications as those of Wheeler,¹ Spaeth,² Jameson³ and Prangen.⁴

The amount of strabismus was determined by the Hirschberg method, in which the position of the corneal light reflex is utilized. Figure 1 is a controlled photographic representation of the position of the corneal reflex as it occurs in the various degrees of deviation. This method was selected because of the simplicity of the determination, and the accuracy of this test correlates the accuracy of the surgical procedures. The factors which introduce variation in this test are omitted from this article. Symmetrically placed corneal reflexes were the criteria used to determine that the eyes were straight. Some of the eyes which were recorded to be straight by this test were slightly eccentrically placed when measured by a telescopic stereoscope. The post-operative results which are presented are not the immediate results but represent the latest records obtainable.

The patients were divided into those with minimum, moderate, marked and maximum grades of strabismus (grades 1, 2, 3 and 4),

1. Wheeler, M. C.: Surgical Treatment of Strabismus: Review of Recent Literature, *Arch. Ophth.* **18**:1000-1010 (Dec.) 1937.

2. Spaeth, E. B.: Principles and Practice of Ophthalmic Surgery, Philadelphia, Lea & Febiger, 1939, chap. 6.

3. Jameson, P. C.: Entity of Muscle Recession: A Short Résumé of Its Technic and Principles with New Supplementary Notes and Illustrations, *Arch. Ophth.* **21**:362-370 (Feb.) 1939.

4. Prangen, A. de H.: Surgery of the Rectus Muscles of the Eye: Selection of Operative Procedures by Differential Diagnosis, *Arch. Ophth.* **18**:151-156 (Feb.) 1935.

according to the amount of the deviation. This classification is helpful both clinically and analytically and is as follows:

Grade 1.....	10 to 20 degrees
Grade 2.....	21 to 35 degrees
Grade 3.....	36 to 45 degrees
Grade 4.....	over 45 degrees

The determination of the amount of excessive or diminished excursions of the eyeball in the extremes of the horizontal plane was used to evaluate the functional condition of the four horizontal rectus muscles. The various amounts of excessive and diminished rotation are represented diagrammatically in figure 2. When the eyeball moved nasally farther than normal, the internal rectus muscle was considered to be

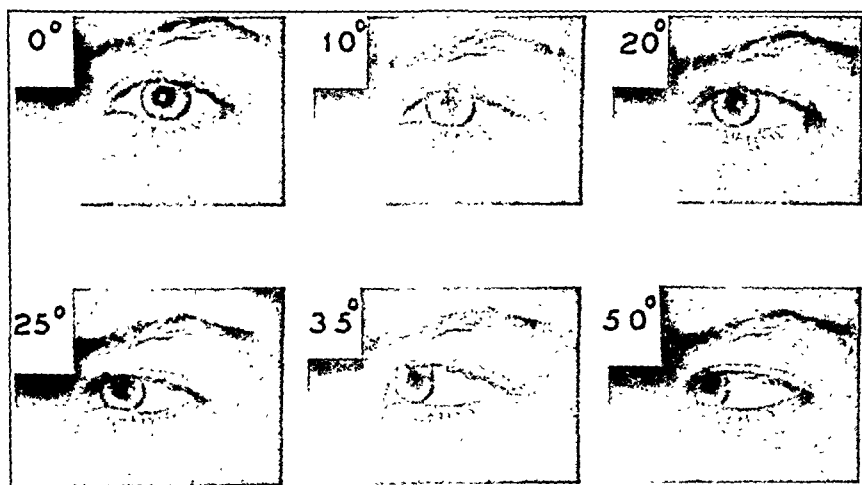


Fig. 1.—The position of the corneal reflex on the deviating eye in the various degrees of strabismus as determined by the Hirschberg method.

functioning excessively. This excessive rotation was classified as +1, +2, +3 or +4, depending on the amount and the speed of the excessive movement of the eye. When the temporal rotation of the eyeball was less than normal, the external rectus muscle was considered to be functioning insufficiently. This insufficiency of rotation was classified as —1, —2, —3 or —4, depending on the degree and the hesitancy of the insufficient rotation. When the motility of the four rectus muscles is properly classified, a comprehensive conception of the abnormal rotations of each eye is obtained. The classification of muscular action is of assistance in deciding, first, which muscles should be selected for operation and, secondly, the distribution of the operative correction which is required. This information makes it possible to operate in accordance with the fundamental principle of determining the amount of correction and the type of procedure by the underlying pathologic muscular condition present in the individual case.

The preoperative and the postoperative distribution of the number of cases of strabismus and of the amount of deviation present in the group in which bilateral recession was done is graphically represented in figure 3. From this graph it is apparent that the majority of patients obtained satisfactory results. It further shows that certain patients were materially improved but did not obtain complete correction and that a smaller group obtained overcorrection. A similar distribution is shown in figure 4, which represents the results of the recession-resection technic.

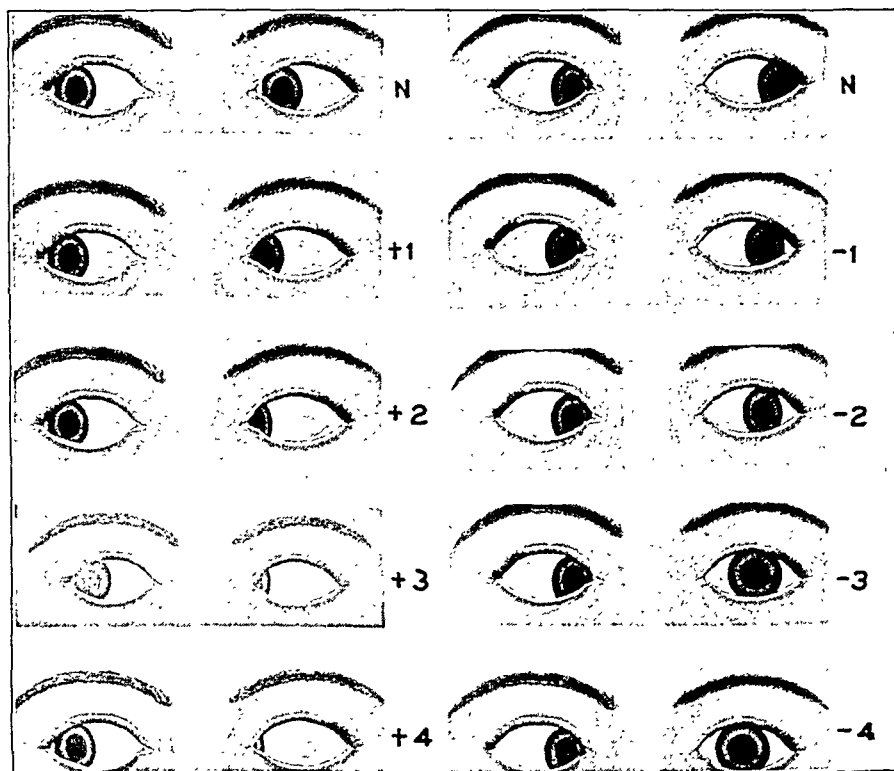


Fig. 2.—The first column shows the normal position of the right eye in eyes right. The left eye is shown in the four grades of hyperaction of the left internal rectus muscle. The second column shows the normal position of the right eye in eyes left. The left eye is shown in the four grades of insufficiency of the left external rectus muscle.

The distribution of the results according to the grade of the strabismus (table 1) shows that patients with strabismus of grade 1 responded better when operated on by the bilateral recession technic than by the recession-resection procedure. Those with strabismus of grade 4 responded more satisfactorily to recession and resection than to bilateral recession. Overcorrection occurred only in cases of strabismus of grades 1 and 2.

The distribution of the results according to the visual acuity (table 2) reveals that the patients with amblyopia ex anopsia responded equally well when operated on by either procedure. The amblyopic patients

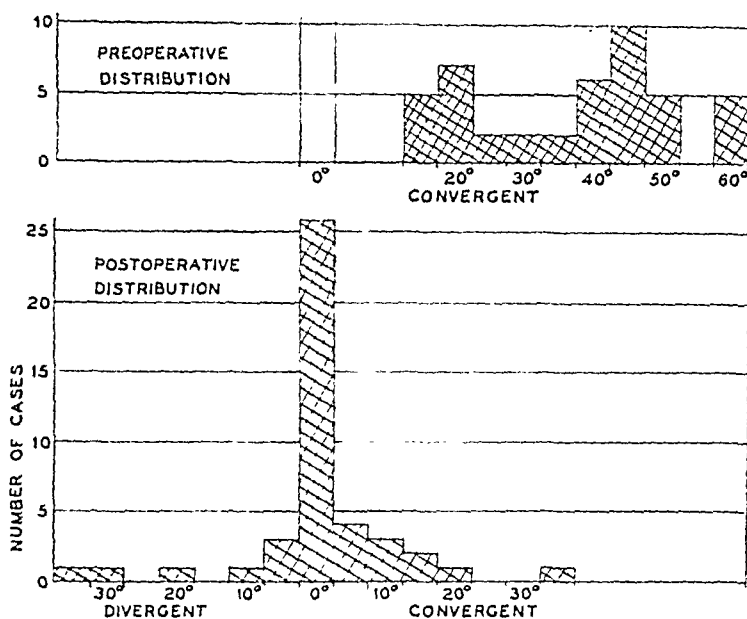


Fig. 3.—Graph showing the distribution of the number of cases of strabismus and the amount of deviation before and after bilateral recession in a group of 44 cases.

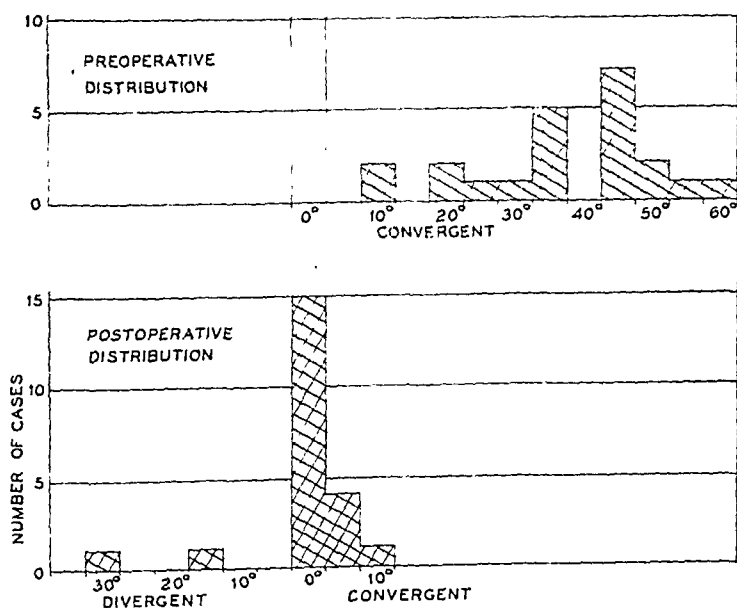


Fig. 4.—Graph showing the distribution of the number of cases of strabismus and the amount of deviation before and after recession combined with resection in a group of 22 cases.

attained a higher percentage of correction than was attained by persons with good vision in each eye.

The amount of operative correction which produced ocular parallelism most frequently in the various degrees of strabismus is given in figure 5. This graph was derived by determining the amount of total operative correction which most frequently resulted in correction for each of the various degrees of strabismus. The graph illustrates four main grades of operative procedures which are applicable to the four corresponding grades of strabismus. Patients with strabismus of 10 to 20 degrees did best when subjected to from 4 to 6 mm. of operative correction, divided between the two muscles. Patients with strabismus of 25 to 35 degrees responded more satisfactorily when they received 6 to 8 mm. of total operative correction, while those with 40 to 50 degrees of

TABLE 1.—*Distribution of the Results According to the Grade of Strabismus*

Result	Recession-Resection					Bilateral Recession				
	Grade 1	Grade 2	Grade 3	Grade 4	Total Cases	Grade 1	Grade 2	Grade 3	Grade 4	Total Cases
Straight.....	1	6	4	4	15	5	5	10	6	26
Undercorrection.....	1	1	3	0	5	1	2	4	4	11
Overcorrection.....	2	0	0	0	2	2	4	1	0	7
Total.....	4	7	7	4	22	8	11	15	10	44

TABLE 2.—*Distribution of the Results According to Visual Acuity*

Result	Recession-Resection		Bilateral Recession	
	6/10 + O. U.	Amblyopia	6/10 + O. U.	Amblyopia
Straight.....	2	11	12	10
Undercorrection.....	1	4	6	2
Overcorrection.....	0	2	1	1
Total.....	3	17	19	13

convergent strabismus attained correction more frequently following 9 to 10 mm. of total operative correction. Twelve millimeters of total correction, divided between the two muscles was necessary to overcome a convergent strabismus of more than 50 degrees. Under identical conditions more correction is usually attained by recession-resection operations than by bilateral recession. Table 2 represents the average amount of correction which will be required in the usual case. This amount of correction will not always produce parallelism, as there are exceptions to these general principles. The reason for the variability in the results is that the effect of the procedure is determined not only by the amount done and by the condition of the muscle on which operation is performed but by the functional condition of the other three horizontal rectus muscles (and occasionally by the condition of the muscles which play a secondary part in the horizontal movement).

It is important to compare the relative rotational power of the muscles of the two eyes. The two main distributions of abnormal rotation encountered were (1) those in which there was an equal amount of rotational disturbance in each eye and (2) those in which there was a unilateral preponderance of abnormal rotation. If the abnormal rotation is equal in the two eyes, the problem is different than it is when there is unilateral preponderance of abnormal rotation. When the rotations are essentially normal in one eye and abnormal in the other, the surgical procedures should be confined to the muscles of the abnormally rotating eye. The importance of this principle is illustrated in table 3. From this table it is seen that all 14 patients whose eyes were straightened by bilateral recession had equal excessive action of the internal rectus muscles. This demonstrates the importance of selecting a pro-

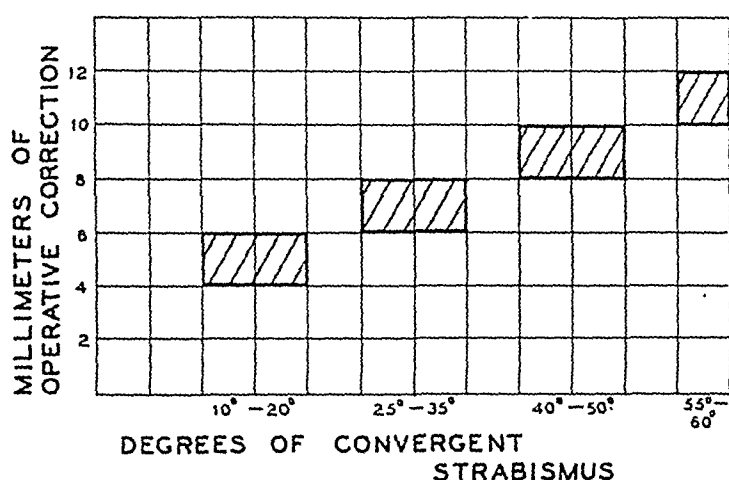


Fig. 5.—Graph showing the amount of total operative correction which succeeded most frequently in the cases of the various degrees of strabismus.

TABLE 3.—*Distribution of the Results According to the Type of Abnormal Muscular Action*

Muscular Abnormality	Recession-Resection			Bilateral Recession		
	Straight	Under-correction	Over-correction	Straight	Under-correction	Over-correction
Unilateral preponderance of abnormal rotations	8	2	0	0	2	1
Equal excess of internal rectus muscles	3	1	0	14	4	2
Equal insufficiency of external rectus muscles	0	0	0	0	2	2

cedure for patients with equally excessive internal rectus muscles which equally diminishes the excessive action of the hyperactive muscle. Those patients who had bilateral excessive action of the internal rectus muscles but whose eyes were not straightened by bilateral recession did not have

the amount of correction shown in figure 5. This demonstrates that not only is it desirable to use the proper procedure but it is also important to graduate the amount of the correction in proportion to the amount of strabismus and the amount of the muscular abnormality.

Likewise, 8 of the 11 patients whose strabismus was corrected by the recession-resection technic had unilateral preponderance of abnormal rotation. This indicates the importance of using a unilateral procedure in cases in which the abnormal rotation is mainly unilateral. Furthermore, patients with bilateral insufficiency of the external rectus muscles and normal rotation of the internal rectus muscles did not respond satisfactorily when subjected to bilateral recession. The rotational abnormalities are mainly due to abnormal innervation of the muscles or to muscular changes, such as hypertrophy, atrophy or contractures or a combination of these, and these factors must be taken into consideration whenever possible. The various causes of these abnormalities are beyond the scope of this analysis.

After the studies are completed, the plans for the operative procedures should be made in the examining room. Occasionally it is necessary to modify these plans on the operating table. The number of millimeters of total operative correction is determined by the amount of the strabismus. If grade 1 strabismus is present, usually a grade 1 operative correction is indicated, and so on for the other grades of strabismus. Exceptions to this rule are those instances in which unusually excessive muscular changes are encountered in the lesser degree of strabismus and in cases of strabismus grade 4 in which there is only slight abnormality of rotation. Smaller amounts of correction are indicated for the same amount of strabismus in children than is necessary for adults. After the number of millimeters of operative correction that should be used is determined, the next decision to be made is the distribution of the correction among the four horizontal rectus muscles. It is seldom desirable to attempt to obtain correction by limiting the operative measures in well established cases to one muscle, and it is rarely necessary to operate on more than two muscles at the time of the first operation. The total operative correction may be divided equally or unequally between the two muscles selected for operation. Since hyperactivity of the internal rectus muscles is usually demonstrable in concomitant convergent strabismus, it is usually necessary to diminish this hyperactivity by recession of one internal rectus muscle or of both of them. If this excessive action of the internal rectus muscles is equal and bilateral, an equal bilateral recession is the procedure of choice. If hyperactivity is unequally present and is not associated with insufficiency of the external rectus muscles, the total amount of recession should be unequally distributed in such a way as to apply most of the correction to the most hyperactive internal rectus muscle. This does not obtain in

cases in which there is over 50 degrees of strabismus, as it is usually necessary to depend on resection combined with recession in cases of grade 4 strabismus. If normal rotation of the internal rectus muscles is present, little if any recession is indicated. In those rather unusual cases in which the main rotational abnormality is an insufficiency of the external rectus muscles associated with fairly normal internal rectus muscles, the main reliance should be placed on resection of the former. The most common type of rotational disturbance encountered was a combination of a hyperactive internal rectus muscle with an insufficient external rectus muscle, and usually this abnormality is much more marked in the muscles of one eye. This is usually the amblyopic eye. The procedure of choice in this type of case is recession of the internal rectus muscle combined with resection of the external rectus muscle of the same eye. If the internal and external rectus muscles are equally abnormal in action, for example a $+2$ internal rectus muscle combined with a -2 external rectus muscle, the total operative correction should be equally divided between the two muscles. If, however, there is a preponderance of hyperaction of the internal rectus muscle, for example a $+3$ internal rectus muscle and a -1 external rectus muscle, the main surgical emphasis should be placed on the hyperactive muscle.

These statements are presented as general principles, because hard and fast rules are not justifiable in such a complicated problem as strabismus and on the basis of such a limited group of cases. In those instances in which the results were unsatisfactory, possible explanations for the outcome were: (1) unusually excessive or diminished muscular action, (2) various congenital ocular defects, (3) previous operations for strabismus and (4) such secondary muscular conditions as hypertrophy, atrophy and contractures.

The final decisions in the individual case should be based on the amount of strabismus, the rotational status of the muscles and the exceptional factors which complicate certain cases. It is most probable that by classification proper evaluation of the functional condition of the muscles, the selection of the appropriate muscles and the graduation of the amount of operative correction that it will be possible to reduce the number of instances in which secondary operations may be required in this group of cases in which the sequelae of the deviation are so well established that all efforts must necessarily fall short of the ideal.

CONCLUSIONS

1. Bilateral recession is ideally applied to young persons with strabismus of grades 1 and 2 in which the rotations show bilateral equal excess of the internal rectus muscles associated with fairly normal external rectus muscles.

2. Recession and resection are most applicable to cases in which there is unilateral preponderance of the muscle abnormalities and for strabismus of grades 3 and 4. It is the method of choice in most cases.

3. The surgical procedure should be directed toward the muscles which are primarily at fault.

4. There are two main surgical causes for unsatisfactory results: (1) undercorrection and overcorrection and (2) failure to select the proper procedure in the given case.

5. Graduated and standard amounts of correction are indicated in the various degrees of strabismus, in the absence of reasons to the contrary.

6. The main reasons to the contrary are: (*a*) the presence of contracture, hypertrophy and atrophy of the muscles; (*b*) unusually excessive or diminished muscular action; (*c*) the presence of the various congenital defects, and (*d*) previous operations for strabismus.

7. No statistical confirmation was available which suggested that the recession-resection technic is more applicable to patients with amblyopic eyes than is bilateral recession.

MEDICAL TREATMENT OF SENILE CATARACT

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In the future more patients with senile cataract will come to the ophthalmologist for treatment. Because of the reduction of infant mortality and the lengthening span of life for those who survive the hazards of infancy, the trend of population in the industrially advanced countries is toward old age. In the United States, over one third of the total population will soon be over 50 years of age, and by 1980 over 14 per cent will be over 65. This trend in the chronologic distribution of the total population by age¹ is clearly shown in the accompanying chart. Along with this general senescence in the population belongs an increased incidence of senile cataract.

The modern treatment of senile cataract is surgical. Operation for cataract in many respects is a radical treatment after the disease has occurred. It is a removal of a part of the eye for which there is no restoration of function without a troublesome artificial substitute. In the surgical treatment of senile cataract the ophthalmologist is in the same position as the orthopedic surgeon is in correcting the osseous deformities of severe rickets. If ophthalmology constantly advances, the treatment will tend to be, first, preventive; secondly, medical, and as a last resort, surgical. Whether or not these objectives will be reached will depend on research and an enthusiastic scientific frame of mind and not on negativism.

In his excellent and remarkable article on "Further Research on Eyes of Monozygotic Twins of Advanced Age with Reference to the Heredity of Senile Symptoms," Vogt² asserted that the chemical studies of the lens are sure to be as futile as the chemical analysis of the optic nerve in Leber's atrophy. Furthermore, in the transactions of the German Ophthalmological Society he³ severely criticized Müller and

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1. Dublin, L. I.: Longevity in Retrospect and in Prospect, in Cowdry, E. V.: Problems of Ageing, Baltimore, Williams & Wilkins Company, 1939, p. 100.

2. Vogt, A.: Weitere Augenstudien an eineiigen Zwillingen höheren Alters über die Vererbung der Altersmerkmale, *Klin. Monatsbl. f. Augenh.* **100**:497, 1938.

3. Vogt, A.: Zur Stardiskussion in Heidelberg, *Klin. Monatsbl. f. Augenh.* **101**:530, 1938.

indirectly all others who are working on the treatment of senile cataract. He stated that because this type of cataract is hereditary it is useless to try to study it chemically. Those who are unfamiliar with the progress in the chemistry of genetics, senescence and the lens may agree with Vogt's point of view. This unqualified attitude is unnecessarily pessimistic in view of the existing data, for there is evidence that senescence can be influenced by various factors.

The contrary opinion is well presented by von Szily,⁴ who said in a recent lecture:

Whether senile cataract, being a hereditary characteristic of old age, will prove amenable to therapy, even if only of a prophylactic nature, remains to be

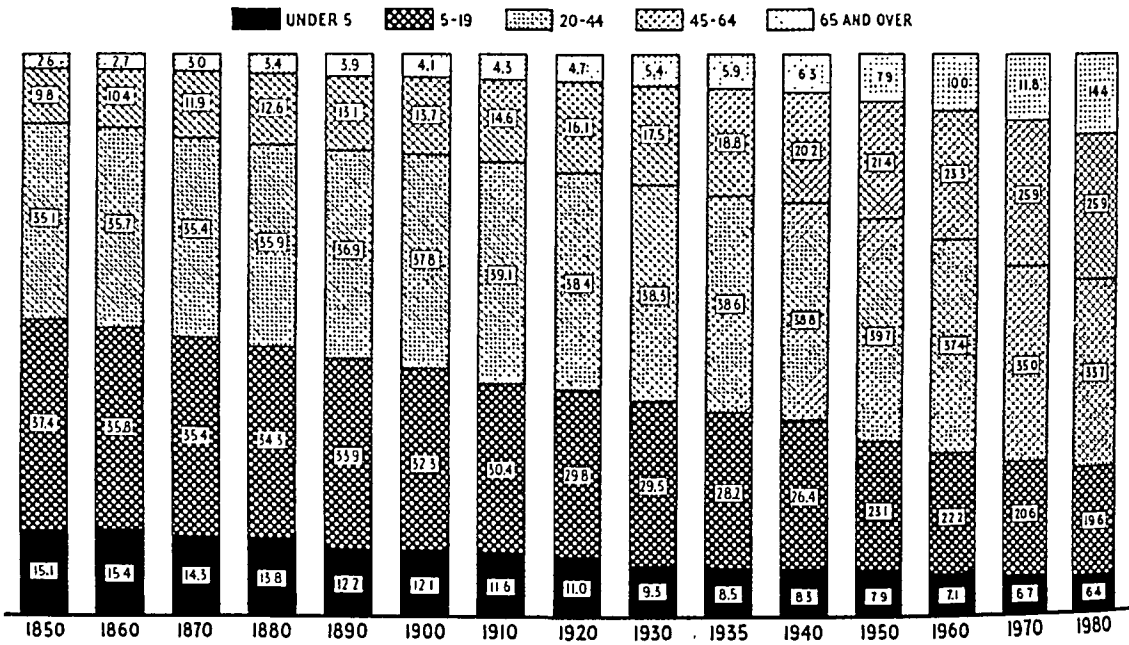


Chart showing the percentage distribution of the total population by age in the United States from 1850 to 1980. (From Dublin, L. I.: Longevity in Retrospect and in Prospect, in Cowdry, E. V.: Problems of Ageing, Baltimore, Williams & Wilkins Company, 1939, p. 100.)

proved. To reject the idea as such, in the sense of a working hypothesis, would seem, however, to be just as much out of place. Who would have imagined only a short time ago, to take just a single instance, that vitamin C, produced from the fruit of the paprika, would at any time be called upon to play such an important part just in the metabolism of the lens? New knowledge always reveals new possibilities, and it is wiser not to lay chains upon the urge for investigation in any direction whatever.

4. von Szily, A.: Doyne Memorial Lecture: The Contribution of Pathological Examinations to the Elucidation of the Problems of Cataract, Tr. Ophth. Soc. U. Kingdom (pt. 2) 58:595, 1938.

The future of medical treatment of senile cataract is not as hopeless as it may appear at the present moment. Since there is good evidence that heredity has an influence on the time factor for lenticular senescence and senile cataract, heredity, senescence and senile cataract may be considered separately. There is no doubt that heredity is a physico-chemical mechanism that may be altered in the laboratory and is altered in nature. Many kinds of changes in heredity have been produced experimentally. There is no need to discuss this point in detail, for it is covered by many treatises. Although the knowledge of the experimental technic in heredity is relatively scanty, an excellent beginning has been made in the modification of heredity. Because there is no convincing experimental work which has been done on the alteration of the heredity of cataract, there is no reason why productive research cannot be done on this problem. Clinically the interest has been active in the observational stage, but experimentally it has been passive. The fact to be emphasized is that heredity is basically chemical in nature and can be changed by man.

To show that some hereditary diseases can be treated, the following examples are chosen as they come to mind, but many others, particularly inborn errors of metabolism, could be added to the list. Owing to a hereditary factor, diabetes mellitus may occur at an early age, but no physician would fail to treat the disease with insulin and a modified diet. Uniovular monozygotic twins may be born with hypothyroidism, which can be corrected with the administration of thyroxin. In xeroderma pigmentosum there is an inborn excess of porphyrins which gives a cutaneous sensitiveness to actinic light. It is fitting to prevent the exposure of this type of presenile disease of the skin to excessive light. There are, of course, hereditary diseases which are not affected by therapy. For example, choroidal coloboma cannot be changed after it occurs, no matter how much research will be done in the future. It is not logical, however, to compare such diseases with senility of the lens.

The time factor of senescence can be modified in the laboratory. MacNider, of the University of North Carolina, has produced premature senility of liver cells. Carrel, at the Rockefeller Institute, has increased the longevity of mice by sheltered life. MacKay, at Cornell University, prolonged the life of rats by restriction of the protein diet. Sherman, at Columbia University, found that calcium, vitamin A and riboflavin (vitamin G) are related to an increased length of life. Simons and Stillman, from the same university, showed that static senile cells can be induced to grow by an unidentified tissue substance. Senescence is therefore not a fixed chemical mechanism. There is good reason to believe that the average person may overcome a part of the biologic senescence by intelligent control of environment, although he

cannot completely avoid the consequences of the hereditary qualities which are born with him.

The biochemistry of the senescent lens has been little studied. Salit is probably the only one who has worked persistently on this problem. Others have been more interested in experimental cataract in young animals, which in most cases is not concerned with senile cataract. A criticism cannot be made on chemical research of the aging lens which is hardly started and which will lag in progress until more is known about senescence, heredity and biochemistry. Such associated fields of research are always interdependent. In the study of senile cataract the application of chemistry and physics is needed to get beyond normal and pathologic anatomy. Physiologic and pathologic senescence are to be differentiated. The effect of infection, intoxication and trauma should be determined, for many senile cataracts may be classified as complicated cataracts. Senescence of the lens may also be a senescence of tissue, which may or may not be a part of the general senescence of the body. The question arises whether or not senile cataract is a primary result of senescence or the secondary response to alterations in the body fluids which have not maintained a proper equilibrium in the aging of the person. Of course there is no possibility of reversing the change which has occurred in a mature or hypermature senile cataract, but there is an expectation that the primary causal factor and the secondary factors which initiate or hasten the maturity of the cataract may be influenced to prevent or retard the cataractous process.

In spite of Vogt's opinion of the hopelessness of the medical treatment of senile cataract, the biochemists are interested in the growth, aging and diseases of the lens and are trying to pass beyond the pathologic changes seen in the tissues and empiric treatment. In speaking of the progress of chemical ophthalmology before the Canadian Ophthalmological Society, Duke-Elder⁵ stated:

. . . but it is again, I think, undergoing a revolution—a revolution less spectacular and dramatic than the previous one because we are delving much more deeply into more subtle, more fundamental, and more difficult things. Hitherto we have been dealing grossly with cause and effect, and have busied ourselves with the structural ruins which disease has left behind; now we are probing more deeply to try to determine the more subtle nature of the initial defect, to pass from the study of structures composed of cells as in 1881 to the study of cells composed of molecules or atoms, to look beyond anatomy and pathology to biophysics and biochemistry.

5. Duke-Elder, S.: *Progress in Ophthalmology*, Canad. M. A. J. **39**:419, 1938.

BILATERAL SYMMETRIC CYSTOID DETACHMENT OF THE RETINA

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Only in the last few years have there been several reports of clinical descriptions of cysts of the retina. These cysts have usually been associated with detachment of the retina. Most of them were solitary and unilateral. Several observers have reported bilateral and symmetric cysts of the retina. Prior to these reports, which will be discussed later, most retinal cysts have been seen on pathologic examination of eyes that were removed for one reason or another, usually painful, hard eyes. The present case is of interest because it shows a bilateral symmetric cystlike detachment of the retina in a location which has not been previously observed. Also, the patient had no complaints referable to his condition, and it was seen on routine examination. It is my belief that this condition will be found more frequently if the observer is aware of its existence and if a special search is made for it in certain types of cases.

REPORT OF A CASE

A white man 33 years of age came to the office complaining of poor vision, difficulty in reading and occasional but severe headaches. His previous history was unimportant except for scarlet fever in childhood. He had been wearing glasses for the past twenty-five years, and one physician had prescribed bifocal lenses a year before. There was no history of injury to the eyes.

On examination, the palpebral fissures were seen to be rather small. They measured 26 mm. in length and 8 mm. in width. The patient had marked exophoria under cover for near vision but orthophoria for distant vision. With homatropine hydrobromide cycloplegia, a compound hyperopic astigmatism was noted. The vision in the right eye was 20/200 and was improved to 20/40 with a + 4.50 sphere combined with + 4.50 cylinder at axis 92. In the left eye the vision was 16/200 and was improved to 20/70 with a + 4.25 sphere combined with a + 5.00 cylinder at axis 95. The cornea was slightly smaller than normal. In the right eye it measured 10 mm. vertically and 10.5 horizontally. The left cornea measured 10 mm. vertically and 11 mm. horizontally. The media were clear; the tension was normal. On examination of the fundi, the disks were seen to be small and reddish, with some indistinctness of the margins. The maculas were normal. The arteries were slightly tortuous, and the veins were normal. In each eye in the extreme periphery of the lower field at 6 o'clock near the ora serrata, a spherical,

Read before the Section of Ophthalmology of the New York Academy of Medicine, Feb. 20, 1939.

sharply defined elevation of the retina was noted. When looked at by indirect illumination, the outlines of the cystlike detachments could be well seen. They had a sharp margin and were distinctly spherical. The extreme lower boundary, however, was beyond the line of vision of the observer and could not be made out. The color was grayish pink, and the surface was slightly irregular and pitted, comparable to the skin of an orange. Several whitish lines were seen on the surface. When looked at by direct illumination with a + 10 sphere in the ophthalmoscope, the detachments appeared to be semitransparent, so that the choroidal vessels could be made out and the margins were much less distinct. In the left eye the termination of a small retinal vessel could be seen coming over the margin of the cyst and terminating in a whitish line. Another interesting finding in the fundus was the appearance of the retina in certain areas. There were numerous bright reflexes not usually found. This has been described by Treacher Collins¹ as a watered silk retina, observed frequently in persons with high hyperopia.

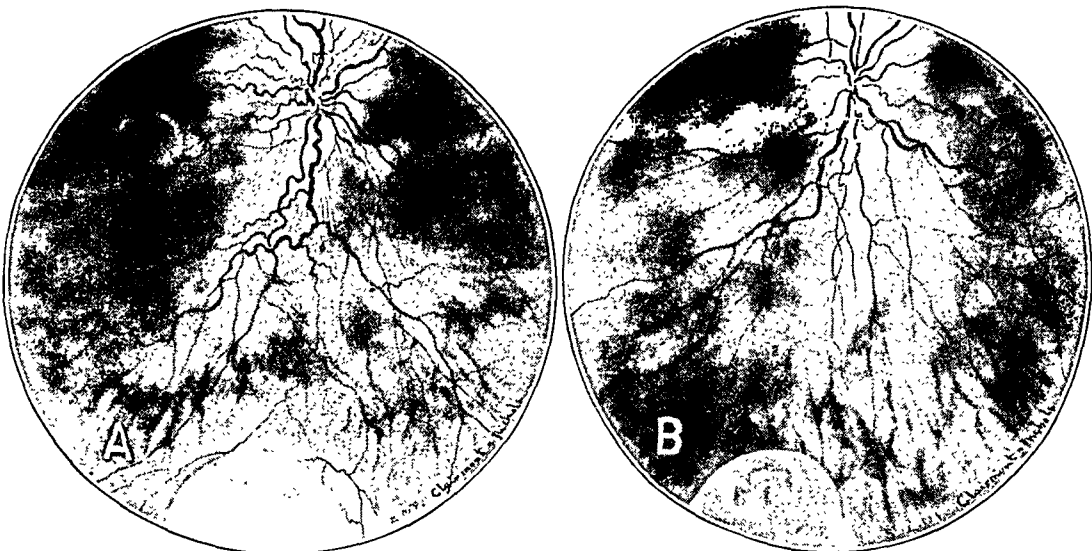


Fig. 1.—*A*, cystoid detachment of the retina of the right eye at 6 o'clock, observed by indirect illumination. This is a photograph of a colored drawing of the fundus. *B*, cystoid detachment of the retina of the left eye at 6 o'clock, observed by direct illumination. The somewhat transparent character is brought out. The choroidal vessels can be seen shining through the cyst.

The rest of the retina appeared to be normal. The fields of vision were taken, and a small defect in the upper field, corresponding to the detached area, was noted in each eye. The patient has been seen several times since the first visit on Dec. 12, 1938. At the time of writing, six months later, there had been absolutely no change in the size, shape or appearance of the cysts.

REVIEW OF LITERATURE

This unusual finding of bilateral symmetric cystoid detachment of the retina prompted a study of the literature. The most comprehensive

1. Collins, E. T., and Mayou, M. S.: *Pathology and Bacteriology of the Eye*, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1925.

recent contribution is an article by Weve.² He cited 10 cases of cysts of the retina associated with detachment. The cysts are usually unilateral and located in the lower temporal field. They are always associated sooner or later with a clinically manifest detachment of the retina. Weve's hypothesis is that the cyst is always in the region of the tear of the retina and may be responsible for the tear. Case 5 of this group is that of a man of 64 with bilateral symmetric cystlike detachment of the retina in the lower temporal region. The patient had hyperopia of 1.5 diopters in each eye. The vision had suddenly become poor and for this reason the patient sought medical aid. The cysts were spherical and transparent. After rupture of the cyst in one eye by a single diathermic puncture, the retina flattened out and the vision improved from 0.1 to 0.5. The other eye was treated in the same

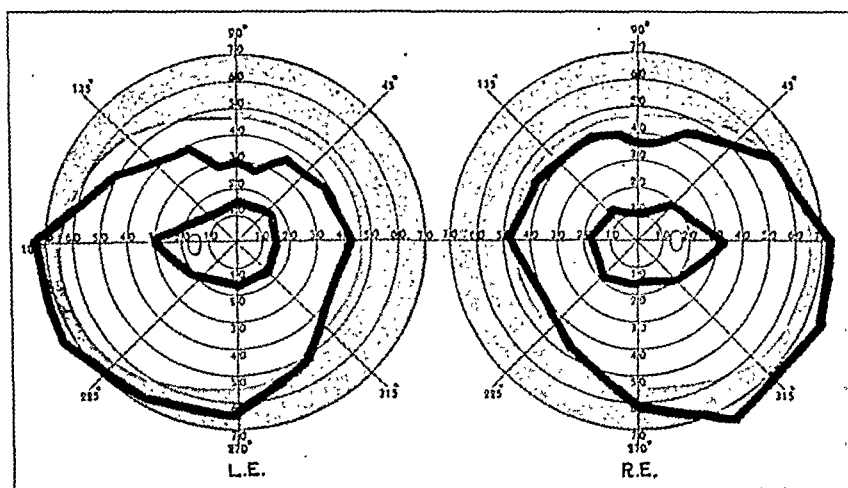


Fig. 2.—Peripheral fields of vision of the patient. Notice the small defect in the upper field. The inner black line is the field of vision for a 3 mm. red object. The form fields were taken with a 3 mm. white object at a distance of 300 mm.

manner, and there was improvement of vision from 0.5 to 1.0. Of interest in this connection is the fact that the field defects noted before operation did not improve afterward. According to Weve, this unexpected result seemed to prove the cystlike nature of the detachments. Judging from the pictures and the field defects, the cystic detachments in this case were much larger than those seen in my case.

Ridley³ reported 5 cases of retinal cysts, in 2 of which the condition was bilateral. In 1 of these cases the patient, a man aged 64, complained of flashes of light and the presence of shadows in the visual field. On examination, retinal cysts were found in each eye in the lower temporal

2. Weve, H.: Die Beziehungen zwischen den grösseren isolierten Netzhautcysten und Netzhautablösung, *Arch. f. Augenh.* **109**:49, 1935.

3. Ridley, H.: Some Practical Points in the Treatment of Simple Detachment of the Retina, *Brit. J. Ophth.* **19**:101 (Feb.) 1935.

quadrant. In the other case the patient, a man aged 57, complained of shadows in the upper nasal field of the right eye. Examination revealed a large cyst in the lower temporal field. A little while later a cyst was noted in the lower temporal field of the left eye and a recurrence in the right. Ridley expressed the belief that the detachment was in the nuclear and reticular layers of the retina rather than between the pigment epithelium and the retina. After operation there was no improvement in the field of vision.

There are several reports in the literature of cysts of the retina which have been observed to form while the patient was under observation for

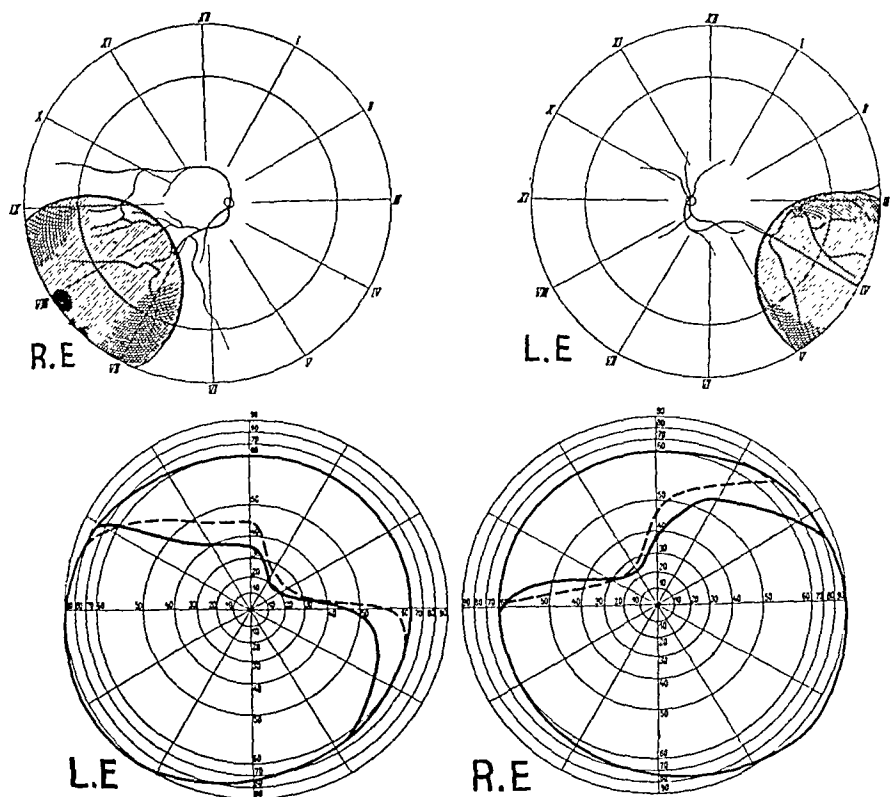


Fig. 3.—Illustrations taken from the report on cysts of the retina with detachment by H. Weve. These cysts are apparently much larger than those seen in my case. (From *Arch. f. Augenh.* 109:49, 1935.)

some other condition. Veil and Guillaumat⁴ reported a case of a young woman with an old iridocyclitis of unknown origin in the right eye which showed a bound down pupil and secondary glaucoma with cupping of the disk. While the patient was under observation a large retinal cyst developed in the left eye in the lower temporal region. There was no sign of detachment or tear. The cyst ruptured spontaneously and left

4. Veil, P., and Guillaumat, L.: Les kystes retiniens, *Arch. d'opht.* 2:977 (Nov.) 1938.

as a residue a pigmented line of demarcation, showing the limits of the cyst.

Fuchs⁵ reported a case in which the patient was observed by his father, E. Fuchs, from 1890 to 1925. She had chronic simple glaucoma in both eyes and was operated on. The cystic area was observed to form in the left eye in the upper outer quadrant over a period of five weeks. A similar cyst appeared temporally in the right eye eight years later. The cyst in the left eye gradually disappeared, and vision became worse because of the glaucoma. The cyst in the right eye could not be followed because it was covered by a membrane secondary to a hemorrhage. Fuchs also reported a second case, that of a man of 57, who had a cystlike detachment of the retina. It was sharply demarcated, transparent and situated in the midperiphery temporally. The retina beyond the cyst, which could be seen, appeared normal.

Van der Hoeve⁶ mentioned a case of cyst of the papilla in which the cyst emptied itself into the vitreous and then refilled.

Rauh⁷ reported a case of cystic detachment at the macula due to exposure to a strong glaring light. The cyst was elevated 2 diopters.

Deutschmann⁸ reported a case of cyst of the retina in which the cyst was located near the ora serrata and appeared and disappeared while the patient was under observation. One eye was removed because the cystic detachment was believed to be due to a tumor. The patient had emphysema and polycythemia. Both factors favor venous stasis.

These 6 cases would appear to show that cysts of the retina may form spontaneously and rupture spontaneously.

There is a group of cases which is of interest because one could suspect the presence of a bilateral symmetric cystic formation in the retina even though the cysts themselves were not seen. This is particularly true of 2 cases reported by Schmelzer.⁹ He described 2 cases in brothers, aged 25 and 32, both of whom had bilateral detachment of the lower half of the fundus with a tear at the ora serrata in the lower temporal region. There was no history of injury or birth trauma. The eyes were emmetropic or slightly hyperopic. The patients were healthy in all respects. Schmelzer agreed with Weve that a primary solitary

5. Fuchs, A.: Ueber Netzhautzysten und über die Entstehung von Netzhautlöchern, *Klin. Monatsbl. f. Augenh.* **98**:145 (Feb.) 1937.

6. Van der Hoeve, J.: Doyne Memorial Lecture: Eye Symptoms in Phakomatoses, *Tr. Ophth. Soc. U. Kingdom* **52**:380, 1932.

7. Rauh, F.: Ein eigenartiger Fall von Veränderung der Netzhautmitte, *Ztschr. f. Augenh.* **63**:48 (Sept.) 1927.

8. Deutschmann, F.: Ueber rezidivierende Zystenbildung in der Netzhaut, *Beitr. z. Augenh.* **9**:591 (April) 1914.

9. Schmelzer, H.: Doppelseitige Netzhautablösung mit symmetrischem Orariss bei zwei nicht kurzsichtigen jugendlichen Brüdern, *Klin. Monatsbl. f. Augenh.* **96**:19 (Jan.) 1936.

retinal cyst may have been the forerunner of the tear and detachment, the cyst being due to a maldevelopment of the retina.

Jancke¹⁰ also reported the case of a man of 21 with a cyst in the macular region of the right eye and another cyst in the lower temporal field with a tear at the ora serrata in the same eye. There was a flat detachment in the lower temporal field of the left eye with a small tear at the ora serrata. The retina also showed a pigment ring surrounding the tear in this eye. This would seem to indicate the probable existence of a cyst in this region previous to the detachment and tear. The correction for the right eye consisted of a + 3.00 sphere combined with a + 4.00 cylinder at axis 90; that for the left eye was a + 1/50 sphere. The right eye had secondary glaucoma.

Vom Hofe¹¹ reported a case of symmetric bilateral retinal detachment in the lower temporal field.

This group of cases, in all of which there was bilateral symmetric retinal detachment in the lower temporal field, seems to fit in with the findings of Weve, who stated that all these detachments are due to the probable presence of a cyst in that region.

Previous to the aforementioned cases, cysts of the retina were seen invariably in eyes that had been removed for some other condition. Of especial interest in this connection is an article by Fuchs,¹² who reported 99 cases of cysts of the retina. They were all seen on pathologic section and were rarely seen clinically. The cysts were present in eyes with inflammatory disease, detachment of the retina and secondary glaucoma. He divided the cysts into 6 groups as follows: (1) cysts on the outer side of the retina between the retina and the choroid; (2) cysts on the inner side of the retina or in the retinal substance between the inner and the outer limiting membrane; (3) cysts caused by atrophy of the retina; (4) cysts associated with secondary glaucoma without detachment of the retina; (5) cysts associated with tumors of the choroid, and (6) cysts associated with detachment of the retina.

Leber¹³ as far back as 1877 saw cysts of the retina on section of pathologic eyes and wondered why they were not seen ophthalmoscopically.

For the sake of completeness, I wish to mention a few other cases that have been reported in the literature of cysts of the retina or

10. Jancke, G.: Echte Netzhautzysten und Netzhautablösung beim Jugendlichen, *Klin. Monatsbl. f. Augenh.* **95**:145 (Aug.) 1935.

11. vom Hofe, K.: Netzhautablösung und Lebensalter, *Klin. Monatsbl. f. Augenh.* **93**:745 (Dec.) 1934.

12. Fuchs, A.: Zur Pathogenese und Anatomie der Netzhautzysten, *Arch. f. Ophth.* **105**:333, 1921.

13. Leber, T.: Die Krankheiten der Netzhaut, in von Graefe, A., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, Wilhelm Engelmann, 1934, vol. 7, pt. 2, p. 1699.

suspected cysts of the retina. Van der Hoeve⁶ mentioned cysts of the retina in phakomatoses. He included in this group Bourneville's disease, the Hippel-Lindau syndrome and Recklinghausen's disease.

Meller and Marburg¹⁴ reported a case of cyst of the retina associated with Czermak-Hippel disease of the retina.

Velhagen¹⁵ reported 2 cases of cysts of the retina, in 1 of which the cyst could be seen with the ophthalmoscope. Both patients had glaucoma, and the eyes had to be enucleated.

Derby¹⁶ reported a case of cyst of the retina on the temporal side of the disk. It was elevated at 6 diopters and was separate from the retinal detachment in the eye.

Gonin¹⁷ had a different theory for the formation of cysts of the retina. In his book on detachment of the retina he has two microscopic sections showing cysts of the external surface of the detached retina. There was a tear at 5 o'clock. He stated that he did not believe that the cysts have any relation to the production of a tear of the retina as a cause. They are more likely to be an effect of the tear. In this connection it is interesting to note that cystic degeneration of the macula is a late sequel of retinal detachment.

Anderson,¹⁸ in a discussion of anterior dialysis of the retina in 1932, mentioned 1 case in which there was a sharp crescentic line near the ora serrata. This line may indicate the presence or the previous existence of a cyst in that region. This article is interesting, because he mentioned four features which are characteristic of cases of the so-called disinsertion or anterior dialysis of the retina.

1. The condition is most commonly seen in the inferior temporal quadrant.
2. The male sex is usually affected.
3. The refraction is nonmyopic.
4. The age of onset is usually much lower than that in other cases of detachment.

Davidson,¹⁹ in the discussion of my paper, mentioned a case of a man aged 35, who was injured in the right eye by a blow from a fist. The first ophthalmologist who saw him reported ecchymosis and hyphemia.

14. Meller, J., and Marburg, O.: Zur Kenntnis des Wesens der sogenannten Czermak-von Hippelschen Netzhauterkrankung, *Ztschr. f. Augenh.* **66**:1 (Sept.) 1928.

15. Velhagen, C.: Ueber Zystenbildung in der Retina, *Klin. Monatsbl. f. Augenh.* **50**:716 (Dec.) 1912.

16. Derby: Retinal Cysts, in Jackson, E.: *Ophthalmic Year-Book for 1911*, Denver, Herrick Book & Stationery Company, 1912, vol. 9, p. 369.

17. Gonin, J.: Die Beziehungen der Retinalzysten zur Netzhautblösung, *Verhandl. d. ausserord. Tag. d. ophth. Gesellsch.* (1921), 1922, p. 273.

18. Anderson, R.: Anterior Dialysis of Retina: Disinsertion or Avulsion at the Ora Serrata, *Brit. J. Ophth.* **16**:641 and 705, 1932.

19. Davidson, M.: Personal communication to the author.

When he was seen by Dr. Davidson several months later, the pupil was found to be larger than the left, but round; a large number of pigment particles were seen in the vitreous and what looked like a giant retinal tear in the lower field near the equator. It measured about 6 disk diameters in length and 1 disk diameter at its widest, in the center. It was elliptic in form, and the edges appeared everted. At each end vessels could be seen bridging the gap. The patient has been followed for five and a half years and during this time has presented the same appearance as when first seen. The vision is 20/20—2. Davidson expressed the belief that there is a rupture of the retina, but the size and the stationary condition led him to suspect that there was originally a retinal cyst which had been ruptured by the contusion.

I have been particularly careful to give as comprehensive a review of the literature as I possibly could, because I feel that this type of case

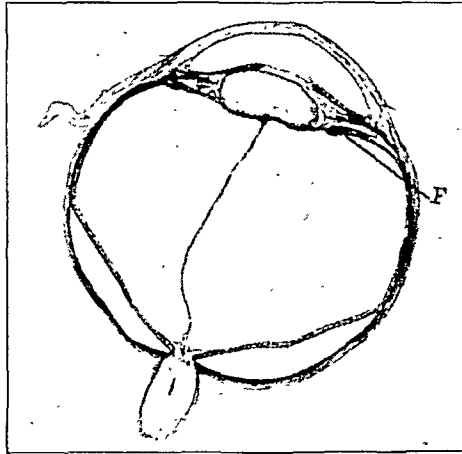


Fig. 4.—A retinal fold at the ora serrata in a microphthalmic eye. (From Ida Mann's "Developmental Abnormalities of the Eye." The illustration is published through the courtesy of the *British Journal of Ophthalmology* and the Cambridge University Press.)

forms a distinct group. It is more than a coincidence that so many of these patients with detachment of the retina in the lower temporal quadrant, occasionally bilateral, are men, usually between the ages of 20 and 40. It would seem to show that there is a group of patients, hyperopic as a rule, who have a predisposition to the formation of cysts, detachments and tears in this region.

ETIOLOGY

The question of the causation of these cysts is of great interest. Weve² in his report gave two possibilities:

1. The cyst may be due to an injury, however slight, which the patient does not even remember. The lower temporal location is a favorite site for contusion of the eyeball.

2. The second possibility is that it is a congenital anomaly. The lower part of the retina is phylogenetically a later developed part of the eye and hence more prone to show abnormalities. The symmetric arrangement in the cases with bilateral cysts cannot be explained on any other basis but that of a developmental abnormality.

I am inclined to favor the latter theory for the reasons given and for several other reasons. The eyes of my patient are what one would call relatively small. In such eyes the inner or retinal layer of the secondary optic vesicle is more readily thrown into folds. Folds of the retina in early embryologic life are common because of the rapid growth of the retinal layer in the first three months. As the eyeball enlarges, the retina is flattened out against the growing and expanding sclera and vitreous. However, in a small eye there might be some areas where this coaptation would not be complete. Such folds have been seen in microphthalmic eyes, and a likely place for them to form is in the region of the ora serrata, which is just before the pars planum of the pars ciliaris retinae. Ida Mann²⁰ has a section of a microphthalmic eye which demonstrates this fact.

Another possibility for the causation of these cysts is that they may be enlargements of the small degenerative cysts of Ivanoff, which are known to occur, especially near the ora serrata. Sometimes they get to be quite large, but rarely, if ever, are they large enough to be seen ophthalmoscopically. They usually are small and microscopic in size.

COMMENT

I made an attempt to determine whether other members of my patient's family were similarly afflicted. Two sisters and one brother were seen. Strangely enough, all these patients had peculiarities of their eyes, but none of them had any cysts. One sister had right hyperesotropia and indistinct margins of the optic disk. Another sister had a marked compound hyperopic astigmatism but no muscle imbalance. The brother had a left esotropia and amblyopia ex anopsia. All were hyperopic.

The question of treatment was then approached. Should an attempt be made to remove these cysts by diathermic puncture as Weve and Ridley have done or should they be left alone? In view of the possibility of detachment of the retina in the future, this is a pertinent question. However, it was decided to wait and watch the patient, chiefly because nothing could be promised to him as a result of the operation, since he had no symptoms referable to his condition. If it is a cyst, what would the contents be? Naturally, this is difficult to determine. Whatever it

20. Mann, I.: *The Development of the Human Eye*, New York, The Macmillan Company, 1928.

is, it must be a highly transparent liquid like the aqueous because of the fact that the cysts are semitransparent.

I am sure that there must be similar cases, but because of the extreme peripheral position of this cystic area and because of the lack of complaints referable to it they are probably overlooked. However, a careful examination of all patients with marked hyperopia might reveal many more similar cases.

SUMMARY

A case of bilateral and symmetric cystoid detachment of the retina in a white man of 33 is reported. The condition was found on a routine examination of the eyes, and there were no symptoms referable to it. The patient had marked compound hyperopic astigmatism. These cysts are believed to be of congenital origin, and the relatively small eye of the patient favors their formation. Similar cases in the literature are grouped together, and the possibility of a syndrome is mentioned. This syndrome would include moderate to marked hyperopia and cystic formation in the lower field of the retina, occasionally bilateral, in young men.

NECROSCLERITIS NODOSA* ASSOCIATED WITH CHRONIC RHEUMATOID POLYARTHRITIS

REPORT OF CASE

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In 1934 van der Hoeve¹ reported 4 cases of a new disease which he had named scleromalacia perforans. Two of the cases were his own and the other 2 belonged to Rochat, who had independently named the condition scleritis necroticans.

Scleromalacia perforans was described as a degenerative disease, accompanied by little or no inflammation and characterized by the appearance of multiple holes in the sclera. Some of the holes were covered by conjunctiva. Others exposed bare uvea to the outside. Adjacent holes often coalesced. The patients in all 4 cases were elderly. Three had advanced chronic rheumatic polyarthritis. One never had had any symptoms of arthritis. Van der Hoeve also mentioned that he had heard of the existence of 2 more cases of this disease, although he had not seen either of the patients. Both were reported as being elderly, and 1 suffered from chronic arthritis.

Apparently all 4 patients were brought to van der Hoeve after the holes had formed. He did not see the condition in its earlier stages. The oculist who referred 1 of the patients stated that one of the holes had been preceded by a yellowish subconjunctival excrescence which had looked like an abscess. When the yellowish excrescence was punctured, a kind of detritus came out.

Van der Hoeve cut off tiny fragments from the walls of some of the holes. Examination revealed "degeneration or chronic inflammation."

The condition in 1 case terminated in opaque corneas and a large hypopyon in each anterior chamber. The latter development was ascribed to infection through the bare uvea.

From St. Luke's Hospital, medical service of Dr. L. W. Frissell and ophthalmic service of Dr. W. Guernsey Frey Jr.

* The name necroscleritis nodosa was suggested by Verhoeff and King.⁵ This name, or Rochat's name, scleritis necroticans, seems much more appropriate for the condition in the case here reported than does van der Hoeve's name, scleromalacia perforans.

1. van der Hoeve, J.: Scleromalacia Perforans, Arch. Ophth. 11:111 (Jan.) 1934.

In the fourth case, according to the history reported to van der Hoeve, attacks of pain and redness had occurred almost once a week for thirty years. This history, the fact that van der Hoeve saw the condition in these cases in the end stages and subsequent case reports by other authors mentioning inflammatory signs all serve to cast some doubt on van der Hoeve's statement that inflammatory signs do not characterize this disease. Very likely it can occur in forms of varying severity.

In 1934 Eber² reported the development of a small spontaneous subconjunctival hole in the sclera, at the limbus, in a 65 year old man who had suffered from chronic arthritis since the age of 10. The hole had penetrated into the anterior chamber, thereby giving rise to a filtering subconjunctival bleb. No inflammatory signs were present when Eber saw the patient.

In 1937 Oast³ reported the case of a man of 76. He had no evidence of arthritis. There first appeared a slightly painful, red, deeply seated nodule with a yellowish necrotic center which looked like an abscess. Incision revealed caseous material. After a few weeks the nodule absorbed and left the uvea exposed beneath the conjunctiva. Subsequent nodules appeared and repeated the same cycle. Some of the holes coalesced. Only one eye was involved. The cornea remained clear, but the vitreous became filled with opacities. All the usual laboratory tests gave negative results. Biopsy showed granulation tissue and cellular debris. Inoculation of the caseous material into the eyes of rabbits failed to show tuberculosis. Cultures for fungi and anaerobic bacteria were negative.

Kiehle⁴ in 1937 reported the case of a 64 year old woman who had suffered since the age of 21 from chronic recurring deforming polyarthritis. Over a period of seven years the sclera of both eyes had gradually lost its normal whiteness and looked slate gray because a thinning of its walls allowed the choroid to show through. The episcleral tissue throughout, after a stage of edematous swelling, had been absorbed completely. When the patient was first seen the corneas and media were clear. At the time of the report both lenses were cataractous. The left cornea had become clouded. Both irides showed multiple radiating perforations. Burning and irritation, but no pain, were present.

In 1938 Verhoeff and King⁵ reported a case in which enucleation was eventually done. The patient, a 52 year old man, had been

2. Eber, C. T.: *Fistula at Limbus (Scleromalacia Perforans)*, *Am. J. Ophth.* **17**:921 (Oct.) 1934.

3. Oast, S. P.: *Scleromalacia Perforans*, *Arch. Ophth.* **17**:698 (April) 1937.

4. Kiehle, F. A.: *Scleromalacia*, *Am. J. Ophth.* **20**:565 (June) 1937.

5. Verhoeff, F. H., and King, M. J.: *Scleromalacia Perforans*, *Arch. Ophth.* **20**:1013 (Dec.) 1938.

afflicted with advanced rheumatoid arthritis for eleven years. When he was first seen he gave a history of redness and soreness in one eye of one year's duration and tumors of three months' duration. The eye was found to have five nodules in the anterior portion of the sclera, with congestion of the overlying conjunctiva.

When a specimen was taken for biopsy 3 drops of thick yellowish pus escaped. Smears revealed pus cells and occasional plasma cells but no bacteria or fungi. All cultures, including those for anaerobes and fungi, were negative. Inoculation of animals failed to show tuberculosis.

Eight weeks after the biopsy specimen was removed the nodule had disappeared and was replaced by a circular hole in the sclera. However, the eye became increasingly painful and congested, and since it was blind from cystoid degeneration of the macula it was enucleated at the patient's request.

Histologic examination showed the disease to be limited to the anterior part of the sclera. The scleral nodules were abscesses consisting of central masses of necrotic pus cells containing fragments of necrotic scleral tissue and surrounded by walls of epithelioid cells. Beyond the walls of epithelioid cells the scleral clefts were packed with plasma cells and fibroblasts. In places the underlying uvea also was involved. Its stroma was largely replaced by new fibrous tissue, chronic inflammatory cells and fibroblasts. The scleral hole showed partial absence of conjunctiva and episclera and replacement of the sclera and uvea by fibrous tissue containing pigment cells. New epithelium formed an external covering.

Verhoeff and King expressed the belief that the pathologic sequence is as follows: First, a small portion of the sclera becomes necrotic. It then quickly becomes walled off by epithelioid cells. Later, pus cells pass through the wall to the necrotic area, which they infiltrate and surround. This causes sequestration and disintegration of the necrotic sclera.

Several subcutaneous nodules from patients ill with rheumatoid arthritis were excised and examined by Verhoeff and King. One nodule showed a large sharply defined area of necrosis. They expressed the belief that the subcutaneous nodules of rheumatoid arthritis and the nodules of scleromalacia perforans are essentially similar in their early stages.

REPORT OF A CASE

On April 5, 1938, a 37 year old white American woman was admitted to the medical service of St. Luke's Hospital, complaining of painful swollen joints of two years' duration, associated with increasing loss of weight and weakness. Also, in the preceding three and a half months both eyeballs had been painful and congested.

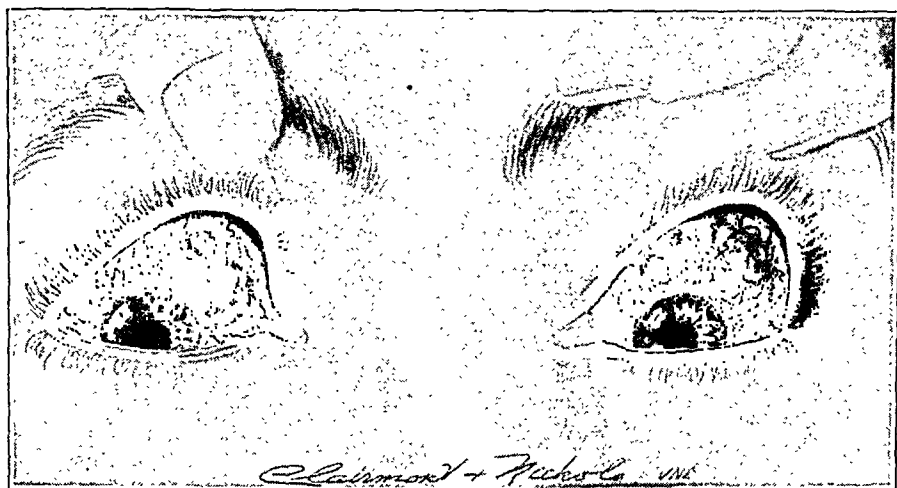
Physical examination revealed severe generalized atrophy of the muscles, contractures of the flexor tendons of the arms and legs, enlargements of the meta-

carphalangeal joints and tenderness of the shoulder, elbow and wrist joints. A diagnosis of rheumatoid arthritis was made.

The patient remained in the hospital for one hundred and sixty-six days, until she died. Emaciation became extreme. Her back and most of the joints of the extremities became unbearably painful and intolerant of any motion. Large doses of morphine failed to relieve the pain fully. The temperature ranged between 99 and 100 F.

All laboratory tests gave essentially negative results except for a constant elevation in the sedimentation rate to between 100 and 130 mm. per hour. Roentgenograms showed generalized atrophy of the articular cartilages with erosion of some of the bones. The teeth and paranasal sinuses showed no focal infection.

Therapy failed completely. Before the patient's admission to the hospital she had been treated by a competent general practitioner with diathermy and other forms of physical therapy. In the hospital she received, among other things, liver extract,



Appearance of the eyes shortly before biopsy.

ascorbic acid, calcium gluconate, synthetic crystalline vitamin B₁ (betaxin), antihistamine, typhoid vaccine intravenously and cobra venom.

Autopsy did not include examination of the head and revealed chronic atrophic arthritis with terminal acute fibrinous pericarditis and bronchopneumonia. Examination of the distal end of the right humerus showed an irregular thickening and rarefaction of the cortical bone, with a fatty gelatinous marrow. The right knee joint showed thinning of the articular cartilage with complete denudation in some areas and small hemorrhages along the margins of the cartilage. Post-mortem culture of the vitreous of the eye did not produce any growth.

The eyes were examined a few days after the patient's admission. Marked bilateral conjunctival and episcleral congestion were present. Both eyes were highly myopic, with liquid vitreous and the usual myopic changes in the fundus. With her glasses, the patient could read newsprint with each eye.

Gradually, within the next three weeks, three comparatively large swellings—two in the right eye and one in the left—appeared beneath the conjunctiva and apparently in the sclera. Each swelling was composed of from one to three yellowish nodules. The swellings in each eye were limited to the upper anterior portion of the sclera and ranged in area from about one fourth to one half of the corneal area. The congestion in both the superficial and the deep blood vessels became maximal

and remained that way. The eyes were painful, but the pain in the joints was more severe and distracted the patient's attention.

One month after the patient's admission a biopsy was done on the largest of the nodules in the right eye. After the overlying conjunctiva had been incised and undermined, the outer wall of the nodule, which looked like thinned-out edematous sclera, was incised. Several drops of moderately viscid, yellowish brown liquid ran out. A culture was taken. Two small fragments of the outer wall were excised for histologic examination. The inner wall of the abscess cavity also was formed by thinned-out sclera, thus showing the nodules to be intrascleral abscesses. The culture grew a gram-positive rod—probably a contaminant. Before further identification could be made, the culture was inadvertently destroyed. The histologic report stated that the specimen was "inflammatory tissue from the sclera."

The nodule which had been incised and drained formed again. Three months after the patient's admission vision became considerably blurred due to bilateral uveitis. About a month later all vision was lost due to massive bilateral hemorrhages of the vitreous. Shortly before the patient's death sclerosing keratitis developed in the right cornea.

COMMENT

The simultaneous involvement of sclera, bones and joints in this case, and in most of the cases reported under the designation scleromalacia perforans, need not seem strange if one remembers that all three tissues are of mesodermal origin. The mystery lies in the failure to grow any organism on culture. Scleral abscesses and nodules are well known in other diseases and conditions which do not affect the articulations or bones.

Pyogenic metastatic scleritis is not an unknown occurrence. Most instances of this seem to have been caused by staphylococcic emboli which reached the sclera by way of the ciliary arteries. The lesions appear suddenly as localized, tender, acutely inflamed swellings. Abstracts of a few typical reports follow:

Terrien and Favory⁶ called attention to the fact that localized suppuration of the sclera is uncommon in comparison with diffuse inflammation or tenonitis. They reported 3 cases of metastatic scleral abscesses; in 2 the abscesses were in the anterior portion of the sclera and 1 in the posterior portion. In 2 cases a staphylococcus was found. One of these patients had furunculosis. All 3 patients recovered completely. Early evacuation of the pus was recommended. The authors also referred to the classic thesis of Dollfus in 1928, in which the latter reviewed all the literature up to that time. In 90 per cent of the cases the staphylococcus was the causative organism.

Sharkovsky⁷ reported a case of scleral abscess due to metastasis from an infected injury of the inguinal region. Corneal edema and iritis were present. Incision of the scleral abscess led to a cure.

6. Terrien and Favory: Circumscribed Abscess of the Sclera, *Arch. d'ophth.* 46:641 (Nov.) 1929; abstracted, *Arch. Ophth.* 4:112 (July) 1930.

7. Sharkovsky, I. A.: Abscess of the Sclera, *Sovet. vestnik oftal.* 1:442, 1932; abstracted, *Arch. Ophth.* 10:537 (Oct.) 1933.

Hudson⁸ reported the occurrence of a metastatic staphylococcic abscess in the sclera at the upper limbus, developing from a carbuncle of the face. Intense iritis was present. Incision relieved the condition and revealed *Staphylococcus aureus*.

Hulka⁹ reported the occurrence of an abscess in the upper anterior portion of the sclera accompanied by severe iridocyclitis in a patient who had just recovered from lobar pneumonia and whose blood had shown pneumococci. Incision of the scleral nodule allowed yellowish pus to run out. Culture of this showed pneumococci identical with those obtained from the patient's blood a little earlier. The eye was lost because of endophthalmitis.

Inflammatory scleral nodules are known to follow herpes ophthalmicus. Penman¹⁰ in 1931 reviewed the literature and reported 4 cases of his own. According to Penman, the course of a typical case is as follows: Two to three months after an attack of herpes ophthalmicus, one or more dark red lentil-sized nodules appear in the sclera. The overlying conjunctiva is inflamed. Iridocyclitis is present. After several months the nodules absorb and leave sharply defined slate gray areas in the sclera. Penman's 4 patients all were elderly—over 60.

A recent editorial¹¹ in *The Journal of the American Medical Association* is of sufficient interest to be quoted in part. It comments on the recent discovery of an infectious polyarthritis in the wild and domestic rats of Java:

. . . The Java pathologists were able to transfer this disease to normal rats by the plantar injection of arthritic joint exudate. . . . The infected joint cavities usually contained a cellular exudate, which was almost invariably sterile on routine bacteriologic test. Filtrates from such exudates were invariably noninfectious. . . . Afterward it was found that a fulminating type of polyarthritis could be produced in normal rats by injecting relatively large doses of arthritic exudates intraperitoneally or intrapleurally. . . . In spite of many careful attempts, it has not yet been possible to cultivate a micro-organism that might be considered to have etiologic significance. Transfer experiments using other than joint tissues obtained from sick rats indicate that the causative agent is not confined to the joints. . . . As yet no instances have been found in which complement fixing, precipitating or neutralizing antibodies have been discovered in the serum of immune rats. This disease presents exceptional features of parallelism with chronic rheumatoid arthritis in man and offers promise of yielding information of practical import.

8. Hudson, A. C.: Staphylococcic Abscess of the Sclerotic, *Arch. Ophth.* **11**: 736 (April) 1934.

9. Hulka, J. S.: Metastatic Pneumococcic Uveoscleritis Following Pneumonia, *Arch. Ophth.* **17**:127 (Jan.) 1937.

10. Penman, G. G.: Scleritis as a Sequel of Herpes Ophthalmicus, *Brit. J. Ophth.* **15**:585 (Oct.) 1931.

11. Infectious Polyarthritis in Rats, editorial, *J. A. M. A.* **113**:148 (July 8) 1939.

THE PLACE OF COATS'S DISEASE AMONG THE DISEASES OF THE RETINA

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The place of Coats's disease, called by Coats retinitis exsudativa (retinitis externa hemorrhagica), among the diseases of the retina is still an uncertain one. The difficulty of characterizing it as a distinct entity is due to the fact that the massive exudations which are characteristic of the disease occur also in the other well characterized diseases of the retina, such as disciform macular degeneration (Junius and Kuhnt) and angiomatosis of the retina (von Hippel), and with extensive retinal hemorrhages of any origin. The difficulty is still heightened by the differences of opinion among investigators as to the cause of the exudations. Coats himself considered them to be the result of the hemorrhages in the retina. He was in doubt about the origin of the hemorrhages but leaned toward the idea that they are the result of some change in the small vessels. Other investigators, following Leber's lead,¹ considered Coats's disease to be a focal inflammatory and necrotizing process in the retina with serofibrinous exudation followed by secondary changes.

A study of the cases reported by Coats and of many of the cases reported by others has convinced me that Coats's disease is a distinct clinical entity; that the exudates and the connective tissue formation in the retina are the consequences of local circulatory disturbances there, beginning with the transudations of plasma and of blood; that the transudations and hemorrhages are the immediate results of peristasis and prestasis and of rupture of small abnormal and dilated vessels; that the small abnormal vessels represent a congenital vascular malformation involving certain branches of the central retinal vessels, and that these vascular malformations find their proper place in a classification of vascular malformations and tumors of the cerebral circulation.

COATS'S DISEASE

Under the heading "Forms of Retinal Diseases with Massive Exudation," Coats² described in 1908 a number of cases clinically and patho-

1. Leber, T.: Die Krankheiten der Netzhaut, in Graefe, A.; Saemisch, E. T., and von Hess, C.: *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 7, p. 1267.

2. Coats, G.: Forms of Retinal Disease with Massive Exudation, *Roy. London Ophth. Hosp. Rep.* **17**:440, 1907-1908.

logically and considered the original change to be in the occurrence of hemorrhages in the deeper layers of the retina. He divided the cases into three groups: (1) those in which there was no coarse vascular disease, (2) those in which marked vascular changes were present and (3) those in which there were arteriovenous communications. In the second communication³ he excluded the last group, which were shown to be cases of angiomatosis retinae. He also entertained doubt as to whether the subdivision into the first two groups could be maintained as they showed more similarities than dissimilarities.

The essentials of the clinical picture are as follows: The disease occurs mostly in young persons and affects only one eye. In only a few cases has the other eye also become affected. The disease progresses slowly and is unnoticed until vision is greatly diminished or completely lost or a divergent strabismus calls attention to the eyes. The cornea, anterior chamber, iris and lens are normal in the early stages; the vitreous may show opacities. Characteristic of the disease is the occurrence of an exudate in the fundus, which is either single or multiple and flat or prominent and is situated in any part of the fundus. The exudate is white or yellowish white and at times gray or of a greenish color. It is situated mainly behind the retinal vessels. Hemorrhages in the retina occur in practically every case, near the veins, on the crest of the exudate or in its periphery. Glistening points and deposits of cholesterol are frequently seen. Deposits of pigment occasionally occur. The disk is usually normal and may be slightly hyperemic.

The retinal vessels present changes in a considerable number of cases, although not in all. These changes are mainly in the small vessels peripheral to the secondary and the tertiary branches and affect the veins more than the arteries. They consist of fusiform and spherical dilatations; loops, twists and tortuosities; the formation of vessels having a brushlike appearance and assuming the form of glomeruli, and of anastomosis between the vessels. The changes in the vessels are usually limited to one particular region of the retina. Coats noted small red areas near the vessels and he had difficulty in deciding whether they were small dilated vessels or hemorrhages.

The course of the disease is usually progressive. Changes occur in the exudate, with retrogression of some of the exudations and the appearance of new ones. Blood exuding from a small dilated vessel results in a large retinal hemorrhage, or it escapes into the vitreous, to be followed later by connective tissue formation (retinitis proliferans). In the later stages detachment of the retina is found; it is localized at first but later involves the whole retina. The end result is partial or

3. Coats, G.: Ueber Retinitis exsudativa (Retinitis haemorrhagica externa), Arch. f. Ophth. 81:275, 1912.

total blindness. The end may be complicated by cataract formation, iritis, glaucoma or softening of the globe.

PATHOLOGIC CHANGES

The characteristic pathologic change is the presence of a mass of connective tissue between the choroid and the retina which may be flat or prominent. This mass is at first loose and cellular, later becoming dense. Lime granules are found here and there and when the choroid is also involved bone formation occasionally occurs. In the center of the connective tissue there is usually a space filled with a detritus consisting of broken-down red corpuscles, leukocytes, cholesterol crystals, phagocytic cells and pigment. The whole gives the picture of an exudate between the choroid and the retina which has become encapsulated with connective tissue.

In the early stages the retina is thickened with a serofibrinous exudate. In the center of the thickened area there is necrosis of retinal tissue involving the outer nuclear layer and the layer of rods and cones and occasionally involving the whole thickness of the retina. At times the serofibrinous layer extends to the inner surface of the retina, but more frequently to the outer surface. The exudate is invaded by phagocytes, the ghost cells of Coats, which according to Leber are derived from the pigment epithelium. Some of these cells are probably histiocytes derived from the adventitia of the blood vessels. In the necrotic areas are found broken-down blood elements, cholesterol and pigment. Later the necrotic areas become encapsulated by connective tissue, which gradually narrows the areas or completely obliterates them. Leber⁴ pointed out that a part of this newly formed connective tissue is seemingly derived from the pigment epithelium, which shows a proliferating tendency outside of the retinal foci. Lamb⁵ observed the changes from pigment epithelium to fibroblasts in cases of exudative retinitis. He found that the connective tissue thus derived was always dense and homogeneous. Internal to the encapsulating focus the retina shows cystic degeneration. Hemorrhages are found in the retina, in the subretinal space and in the vitreous. At first there are no adhesions between the choroid and the retina; however, in the later stages adhesions as well as slight cellular infiltration of the choroid are observed. The lamina vitrea is usually intact. Detachment of the retina is found, with many phagocytes and with coagulated fluid in the subretinal space.

Changes in the vessels were not found in all cases. They are as follows: (1) marked dilatation and congestion, the dilatations being so

4. Leber,¹ p. 1302.

5. Lamb, H. D.: The Pathogenesis of Some Intraocular Osseous Tissue: True Metaplasia in the Eye, *Am. J. Ophth.* **18**:409, 1935.

marked in some cases as to give the appearance of a cavernous angioma; (2) thrombi in various stages of organization in some of the dilated vessels; (3) thickening and hyaline degeneration of the vessel walls; (4) perivascular accumulation of cells in a few cases, and (5) proliferation of the endothelium and loss of staining of their nuclei in a few cases.

ETIOLOGY

Coats's presentation of his clinical and pathologic material and his pathogenic deductions are a masterpiece of clear thinking and exposition. Coats came to the conclusion that the pathologic process is initiated by hemorrhages into the retina derived from the capillaries in the external reticular layer. The extravasated blood either pushes the external limiting membrane outward or causes a rupture and forms a localized or diffuse extravasation. This extravasation of blood is responsible for the destruction of the retinal elements, for the serofibrinous fluid in the neighboring parts of the retina and for the cystic degeneration of the retina. The extravasated blood causes a reactive influx of phagocytes and a proliferation of connective tissue, which encapsulates it. The central part gradually becomes liquefied and is filled with debris of cells and cholesterol. The edema in the adjacent parts of the retina extends into the subretinal space, with resulting detachment of the retina. The reactive process, which resembles a mild inflammatory process, occasionally extends to the choroid, with the formation of adhesions between the retina and the choroid. Coats found no evidence of an inflammatory process and did not consider any such process as an etiologic factor.

The question arose as to what caused the hemorrhages. In an occasional case Coats considered hemorrhage due to a forcible forceps delivery or to a sudden increase in intravascular pressure as a result of whooping cough. However, in the greater number of cases such a causation could not be assumed. Coats considered therefore two possibilities: (1) an abnormal constitution of the blood, which is responsible for the hemorrhage, and (2) a primary change in the vessels, especially the smaller ones. He inclined toward the second of the two possibilities, although in some cases no vascular changes were found. He considered dilatation of the vessels and their increase in number a secondary change.

The clear insight of Coats into this disease incites admiration. It is unfortunate that Leber¹ did not agree with Coats's interpretation of the pathogenesis of the disease and assumed an inflammatory and necrotizing process in the retina as the cause. A number of authors have followed him in this interpretation, although a critical examination of the pathologic changes and of the course of the disease is strictly against it.

A study of the pathologic reports of Coats's disease shows that in spite of the necrosis of the retinal elements, which would argue for a severe inflammation, some of the essential elements of an inflammatory process in the retina are not present. There is no outpouring of leukocytes into the retina and no clouding of the vitreous with inflammatory elements; there is only a mild involvement of the choroid by round cells, and then only in some cases, evidently of a secondary character. The notion that Coats's disease is an inflammatory process of the retina cannot be entertained by Leber's own criteria.⁶

The essential features of the pathologic process of Coats's disease aside from the changes in the vessels are: (1) the outpouring of plasma into the retina; (2) the hemorrhages; (3) the necrosis of the retinal elements; (4) the deposit of fibrin and lipoids among the retinal element; (5) the invasion of the retina by phagocytes, and (6) the organization and encapsulation of the hemorrhages and transudates which are too large to be absorbed. The first and second features represent the immediate result of local circulatory disturbances, while the necrosis is the result of the hemorrhages. Fibrin is deposited by the transuded blood plasma, and the appearance of lipoids is due to the disintegration of blood of necrotic retinal tissue. Invasion by phagocytes and the organization and encapsulation of hemorrhages too large to be absorbed are the further results of local circulatory disturbances. All these pathologic features together represent a good example of the results of local circulatory disturbances, such as I have outlined on another occasion.⁷ The progressive course of the disease and the presence in the retina of lesions in various stages of organization make it necessary to assume that the transudation of plasma and the hemorrhage keep on recurring. The problem involved is as to the character of the vascular changes which permits repeated transudations of plasma and of blood.

IMPORTANCE OF THE DILATED SMALL VESSELS IN THE AFFECTED AREA OF THE RETINA

In discussing local circulatory disturbances⁷ it was mentioned that transudation of plasma and hemorrhages occur whenever there is a certain definite slowing of the blood stream in the terminal vascular units. The slowing occurs whenever there is a constriction of the artery above the dilated terminal units, and the degree of slowing results in peristasis characterized by transudation of plasma or by prestasis characterized by the passage of blood through the capillary walls. To produce repeated transudations of plasma and repeated hemorrhages such as are found in

6. Leber,¹ p. 567.

7. Elwyn, H.: Circulatory Disturbances in the Retina in Arteriosclerosis and Essential Hypertension, *Arch. Ophth.* **21**:775 (May) 1939.

the retina in Coats's disease, the small terminal units, the prearterioles, the capillaries and the venules, must be repeatedly in a state of dilatation either of functional or of organic origin. There is nothing known of any functional constriction of arteries in this disease, nor does it seem possible that any functional constriction of the arteries with dilatation of the terminal units could produce the ophthalmoscopic and pathologic picture of Coats's disease. There is, however, plenty of evidence of an organic dilatation of small vessels in certain areas in the retina in this disease.

In the various pathologic reports of cases of Coats's disease the vascular change most frequently mentioned is dilatation of the small vessels in the affected area of the retina. It is found in most of the reported cases. Coats mentioned it in seven of nine pathologic reports. In case 8 the dilated vessels almost took on the appearance of a cavernous angioma; in the other cases the dilatation of the vessels was not so marked. Berg⁸ reported 4 cases in which there were marked vascular changes, narrowing and dilatation of vessels, thrombi and dissecting aneurysms. His report is accompanied with unusually good colored illustrations depicting the dilatations and aneurysms. Rados⁹ reported a case in which dilated vessels were found. Miyashita and Nisyake¹⁰ reported a case in which the vessels had almost the appearance of a cavernous angioma. There was aneurysmal dilatation of the small vessels, and all the capillaries in the neighborhood were enlarged. In Sattler's¹¹ 2 cases the vessels were markedly dilated, as they were also in Wölflin's¹² case, in ten Doesschate's¹³ 2 cases, in Zinsser's¹⁴ 5 cases, in Marchesani's¹⁵ case and in von Hippel's¹⁶ 2 cases (cases 2

8. Berg, F.: Beitrag zur pathologischen Anatomie der Retinitis exsudativa, Arch. f. Ophth. **98**:211, 1918-1919.

9. Rados, A.: Ueber die Veränderungen im Frühstadium der Retinitis exsudativa, Arch. f. Ophth. **105**:973, 1921.

10. Miyashita, S., and Nisyake, Y.: The Pathological Anatomy of Retinal Degeneration with Multiple Aneurysms, Brit. J. Ophth. **5**:448, 1921.

11. Sattler, H.: Ueber die pathologisch-anatomischen Veränderungen der Retinitis exsudativa (Coats), Klin. Monatsbl. f. Augenh. **74**:222, 1925.

12. Wölflin, E.: Beitrag zur pathologischen Anatomie der Retinitis exsudativa externa, Arch. f. Ophth. **117**:33, 1926.

13. ten Doesschate, G.: Ueber Retinitis exsudativa externa, Klin. Monatsbl. f. Augenh. **79**:505, 1929.

14. Zinsser, F.: Beitrag zur Kenntnis der exsudativen Netzhauterkrankungen (Fünf Fälle von Retinitis exsudativa, ein Fall von Angiomatosis retinae), Arch. f. Ophth. **121**:686, 1929.

15. Marchesani, O.: Zur Anatomie der Angiomatosis retinae und Retinitis exsudativa, Arch. f. Augenh. **103**:643, 1930.

16. von Hippel, E.: Angiomatosis Retinae und Retinitis Exsudativa Coats, Arch. f. Ophth. **127**:27, 1931.

and 3). Lamb¹⁷ reported 8 cases of exudative retinitis. Dilatation of some vessels or the presence of numerous vessels is merely mentioned in his second, third, fifth and seventh cases. He considered the dilatation of the vessels as secondary and the primary condition as inflammatory.

In spite of the few cases in which no dilatation of vessels were reported, it is obvious that dilatation of the vessel and an increase in their number in the affected area of the retina constitutes one of the essential elements in the pathologic picture of Coats's disease. In such dilated vessels a slowing of the blood stream must occur and with it the states of peristasis and prestasis with the accompaniment of transudation of plasma and of blood. In addition, there is rupture of small dilated vessels, as shown by the presence of small dissecting aneurysms in some of the cases. The transudation of plasma and of blood initiates the changes in the retina which altogether constitute Coats's disease.

The numerous small vessels in the affected area of the retina, the aneurysmal dilatation, the dissecting aneurysms and the hyalinized or thickened walls evidently represent vascular malformations or angiomas localized in a certain vascular area of the retina.

In the same year and in the same volume of the *Archiv für Ophthalmologie* in which Coats's second publication³ appeared there was also a contribution by Leber¹⁸ on a form of retinal degeneration characterized by the presence of multiple aneurysms. Leber had 2 cases of his own and had collected 11 more from the literature. He considered these cases as belonging to a group which is partly overlapped by the second group of Coats's cases, the one in which there were marked vascular changes. One of Leber's collected cases is included among Coats's series.

An advance in the understanding of Coats's disease was brought about by the publications of Junius.¹⁹ Junius attempted to correlate Coats's disease and Leber's retinal degeneration with miliary aneurysms with a disease known as "hereditary familial telangiectasia," also known as Osler's disease. In this disease, which has been shown to be familial and hereditary, there occur numerous telangiectases in various parts of the body. Areas of predilection are the skin and the mucous membranes, especially of the nose, with frequent occurrence of epistaxis. Telangiectases occur also in the mucous membrane of the respiratory tract and the digestive and the urogenital tracts as well as in the brain and in the eyes. These telangiectases consist of dilated capillaries and

17. Lamb, H. D.: Exudative Retinitis, *Am. J. Ophth.* **21**:618, 1938.

18. Leber, T.: Ueber eine durch Vorkommen multipler Miliaraneurysmen charakterisierte Form von Retinaldegeneration, *Arch. f. Ophth.* **81**:1, 1912.

19. Junius, P.: Zur Aetiologie der Retinitis exsudativa Coats, *Klin. Monatsbl. f. Augenh.* **92**:748, 1934; Angiomatosis retinae—Retinitis exsudativa Coats—Morbus Osler, *Ztschr. f. Augenh.* **84**:193, 1934.

venules which have a tendency to bleed. The primary disturbance is considered by some investigators to be a defect of the mesenchymal tissue, which fails to develop normal endothelial cells lining the vessels and normal supporting tissue. The condition must be regarded as a system disease. The tendency to bleed is considered a result of the defective endothelial cells. Junius considered the possibility that the miliary aneurysms in the retina might be equivalents of such telangiectases and that they possibly represent a localized weakness in mesenchymal differentiation. The miliary aneurysms in the retina which are associated with retinal degeneration unquestionably belong to the classification of Coats's disease.

The foregoing review leads to the following logical conclusion: The fundamental pathologic factor in Coats's disease is a vascular malformation involving a certain definite area of the retinal circulation. The malformation involves the small vessels, small arteries, capillaries and veins, with the formation of miliary aneurysms, dilatation of the vessels and with defective walls which are subject to hyalinization, thickening or thinning and to rupture. Such malformations must necessarily be of congenital origin. With the years the vessels are likely to dilate more and to rupture, with clotting of the blood. The slowing of the blood flow in these vessels and the occasional ruptures are responsible for the transudation of plasma and for the hemorrhages. The transuded plasma and the hemorrhages in the retina incite the reactive changes, the invasion by phagocytes and the production of connective tissue.

The assumption that a vascular malformation which manifests itself in dilated vessels is the essential factor in Coats's disease leads to the problem whether such a malformation finds a definite place in a classificatory scheme of vascular anomalies. The retinal circulation is a part of the cerebral circulation, and it is therefore of interest to know whether the various vascular malformations in the brain find their counterpart in the retina.

CLASSIFICATION OF VASCULAR MALFORMATIONS AND TUMORS IN THE BRAIN

The classification of vascular malformations and tumors in the brain varies with different investigators. Cushing and Bailey²⁰ divided tumors of the blood vessels into two groups: (1) angiomatous malformations and (2) angioblastomas or true neoplasms of blood vessel elements. Bergstrand, Olivecrona and Tönnis²¹ recently published a most comprehen-

20. Cushing, H., and Bailey, P.: *Tumors Arising from the Bloodvessels of the Brain*, Springfield, Ill., Charles C. Thomas, Publisher, 1928.

21. Bergstrand, H.; Olivecrona, H., and Tönnis, W.: *Gefäßmissbildungen und Gefäßgeschwülste des Gehirns*, Leipzig, Georg Thieme, 1936, chap. 2, pp. 8-68.

sive account of the pathologic picture of the malformations and tumors of the vessels of the brain. They pointed out that it is impossible to draw a sharp line of distinction between the two. All angiomas are primarily malformations, even when they appear as true tumors, such as the Lindau tumors. Bergstrand expressed the opinion that Virchow's classification of angiomas is still the best, and he added to it the forms of angioma which have been discovered since the publication of Virchow's lectures. Virchow²² classified angiomas into two types: (1) angioma cavernosum and (2) angioma racemosum. The first tumor is characterized by blood spaces, between the walls of which there is no foreign tissue. In the second type each vessel forms a separate entity, and the tumor consists of a mass of vessels which are separated from one another by some other tissue. Tumors of this type (angioma racemosum) are divided into four subgroups: (a) telangiectasia, in which the vessels are undoubtedly capillaries but are dilated, with changes in their walls and an increase in their number; (b) angioma racemosum arteriale, in which the arterial branches of a certain definite vascular area are affected; (c) angioma racemosum venosum, in which a group of venous branches make up the vascular tumor, and (d) aneurysma arteriovenosum, dilated arteries and veins, in which there is a direct connection between the two. Bergstrand and his co-workers added another subgroup in which the vascular malformations resemble telangiectasia and involve in the complete syndrome the brain, the choroid of the eye and the skin. They named the syndrome Sturge-Weber disease.

To this classification Bergstrand, Olivecrona and Tönnis added two groups which were not known at the time of Virchow: (1) angio-blastoma or angioreticuloma or Lindau's tumor and (2) angioglioma.

In summary, this classification of angiomas of the brain as outlined by Bergstrand and his co-workers follows:

1. Angioma cavernosum
2. Angioma racemosum
 - (a) Telangiectasia
 - (b) Sturge-Weber disease
 - (c) Angioma racemosum arteriale
 - (d) Angioma racemosum venosum
 - (e) Aneurysma arteriovenosum
3. Angioblastoma, angioreticuloma or Lindau's tumor
4. Angioglioma

This classification gives an adequate account of the vascular malformations and tumors which occur in the brain. I shall discuss briefly

22. Virchow, R.: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1867, vol. 3, p. 460.

the individual forms so far as they also occur in the retina. For didactic purposes, I shall take them up in the reverse order.

Angioglioma.—The term angioglioma was used, according to Bergstrand, by Roussy and Oberling for a tumor which supposedly consists of a combination of malformations of vessels and of nerve tissue. However, the tumor of Roussy and Oberling and similar tumors which Bergstrand examined seemed to him to belong to the group of Lindau tumors. Although these tumors do not belong to a special group, he proposed to retain the term angioglioma for tumors consisting of a combination of vessels and nerve tissue. He considered as the best example of such a tumor the subependymal tumor found in cases of tuberous sclerosis (Bourneville's disease). These consist "on the one hand of large giant astrocytes rich in protoplasm and of piloid astrocytes and on the other hand of a convolute of vessels which for the greater part are calcified."²³ In this monograph²¹ a photographic reproduction is presented of an angioglioma in a case of tuberous sclerosis.

In cases of tuberous sclerosis small mulberry-shaped tumors have been found in the retina; these were first described by van der Hoeve.²⁴ Pathologic examination showed them to consist of nerve fibers and large cells, but the presence of any dilated vessels in them has not been reported. So while angiogliomatous tumors occur in the subependymal regions of the brain in tuberous sclerosis, they have not been reported in the retina in association with this disease.

A tumor consisting of a combination of glial elements and of miliary angiomas is described by Bergmeister²⁵ in a case of microphthalmos with orbital cyst. He characterized the tumor as angioglioma and mentioned a similar case reported by Seefelder.

Angioblastoma or Angioreticuloma.—This type of tumor is also known as Lindau's tumor or as Lindau's disease. When in addition to the involvement of the brain a similar tumor is also found in the retina, the condition is known as Hippel-Lindau disease. In 1926 Lindau²⁶ described these tumors in cerebellar cysts. The tumor has autonomous growth and is of meningoencephalic origin. It consists of angioblasts which form masses of capillaries surrounded by a network of reticulin. Between the capillaries there are endothelial cells filled

23. Bergstrand, Olivecrona and Tönnis,²¹ p. 60.

24. van der Hoeve, J.: Augengeschwülste bei der tuberösen Hirnsklerose (Bourneville), Arch. f. Ophth. **105**:880, 1921.

25. Bergmeister, R.: Ueber gliöse Wucherungen im Auge by Mikrophthalmus congenitus und deren Beziehungen zur Angiomatosis retinae, Arch. f. Ophth. **105**: 1, 1921.

26. Lindau, A.: Studien über Kleinhirnzysten, Acta path. et microbiol. Scandinau., 1926, supp. 1, p. 1.

with fat droplets resembling xanthomatous cells. The tumors occur especially in the cerebellum and also in the spinal cord and in the retina. With such a tumor, which frequently forms cystic spaces, there are also found multiple cysts in the pancreas and less often cysts in the kidneys, hypernephromas and tumors of the epididymis.

In the retina the disease is well known to ophthalmologists as angiomatosis retinae, or Hippel's disease. Here the tumor is a capillary angioblastoma, made up of angioblasts similar to those found in the cerebellum. These cells form capillary spaces and solid cellular cords. They also form cystic spaces and frequently give rise to a secondary proliferation of glia. Transudation of plasma in the retina is a frequent occurrence. Detachment of the retina occurs later.

In the early stages, before complications have set in, the ophthalmoscopic picture is striking. A pair of dilated, enlarged and tortuous vessels, an artery and a vein, course toward a raised roundish tumor in the periphery, which they enter. Several such tumors, each with a pair of dilated vessels, may be seen. Later the picture is complicated by exudates, detachment of the retina, iridocyclitis or glaucoma.

Lindau expressed the opinion that a vascular malformation in the roof of the fourth ventricle is the plausible matrix of the tumor in the cerebellum. The vascular plate is there formed at about the third embryonal month, at the same time that the retina becomes vascularized.

Arteriovenous Aneurysm.—This is a vascular anomaly in which there is an enlargement and dilatation of an artery and the corresponding vein. Both vessels are either directly united by means of one good-sized vessel or of several such vessels, or a bunch of intertwined dilated vessels are interpolated between the artery and the vein instead of the normal capillary bed. Bergstrand made the statement that the vessels which make up the arteriovenous aneurysm are so malformed as to scarcely deserve the name of either artery or vein. The structure of the walls of the vessels resembles that which makes up the walls of the vessels of the angioma racemosum venosum. Numerous cases of arteriovenous aneurysm occurring in the brain have been reported. The literature is found in the publications of Bergstrand and his associates,²¹ Cushing and Bailey,²⁰ Dandy²⁷ and Brock and Dyke.²⁸

In the retina arteriovenous aneurysm is a rare disease. Leber²⁹ mentioned 4 cases from the literature. Stokes,³⁰ who reported a case

27. Dandy, W. E.: Arteriovenous Aneurysm of the Brain, *Arch. Surg.* **17**: 190 (Aug.) 1928.

28. Brock, S., and Dyke, C. G.: Venous and Arteriovenous Angiomas of the Brain, *Bull. Neurol. Inst. New York* **2**:247, 1932.

29. Leber,¹ p. 37.

30. Stokes, W. H.: Racemose Arteriovenous Aneurysm of the Retina (Aneurysma Racemosum Arteriovenosum Retinae), *Arch. Ophth.* **11**:956 (June) 1934.

of his own, found 2 more cases in the literature. The ophthalmoscopic picture is striking. The involved vessels are dilated, enlarged and tortuous, but the arteries and veins can be distinguished and the places of anastomosis can frequently be seen. The disease is perhaps not so rare as one would judge from the few reported cases. At the meeting in May 1939 of the Section of Ophthalmology of the New York Academy of Medicine there was presented a case of arteriovenous aneurysm of the retina. Another case was seen at the New York Eye and Ear Infirmary, and excellent colored photographs were obtained by Dr. D. Bogart.

Angioma Racemosum Venosum.—The venous angioma consists of dilated venous channels. There is a gradual transition between such an angioma and a varix. The growth occurs in the meninges of the brain or in addition also in the brain tissue below the meningeal involvement.³¹ The walls of the vessels involved in the angioma are markedly deformed (Bergstrand, Olivecrona and Tönnis).

In the retina dilated, enlarged and tortuous vessels occasionally occur. They have been described by Leber³² under the heading of congenital tortuosity of retinal vessels. He reproduced in his publication a picture made in one of Nettleship's cases. Dilatation of the retinal veins is also found occasionally in what is now known as Sturge-Weber disease in combination with vascular nevi of the skin. Case 3 of Brock and Dyke is of special interest. This was a case of venous angioma of the brain in which the right eye showed on ophthalmoscopic examination that "the veins were so greatly engorged and tortuous that the disc was almost completely obscured."²⁸ The condition was regarded as a varicosity or venous angioma of the central vein. The angiomatous lesion extended "from the right retina and optic nerve, chiasm, and tract to the right midbrain." The tumor was composed of thin-walled blood vessels.

Angioma Racemosum Arteriale.—Arterial angioma supposedly consists of dilated arteries, the vessels leading to the tumor and draining the blood from the tumor being arterial in character. Bergstrand stated that he considered it doubtful whether such tumors actually occur, and with the walls of the vessels of racemose angioma malformed it would be difficult to characterize them as arteries. Brock and Dyke²⁸ classified their case 8 as one of arteriovenous angioma, although they appended to the histologic description the diagnosis, arterial angioma, made by Orton and Wolf.

31. Bailey, P.: *Intracranial Tumors*, Springfield, Ill., Charles C. Thomas, Publisher, 1933, p. 194. Cushing and Bailey.²⁰ Brock and Dyke.²⁸

32. Leber,¹ p. 53.

Leber³³ mentioned under congenital tortuosity that occasionally the arteries of the retina are also tortuous with the veins. He did not mention any case in which the arteries alone are involved.

Sturge-Weber Disease.—In 1879 the English physician Sturge described a case of congenital glaucoma and naevus flammeus of one side with epileptiform convulsions of the other side of the body. He concluded that the brain must be the seat of a vascular nevus. Vascular nevi of the skin with glaucoma had already been observed before Sturge's report. In 1922 F. Parkes Weber found roentgenographically characteristic parallel lines in the brain in such a case. Since then this syndrome has attracted more attention, and Bergstrand and his associates have proposed the name Sturge-Weber disease for it.³⁴ In the complete syndrome there is found clinically vascular nevi of the skin, glaucoma, convulsions and frequently paresis and idiocy. However, the complete syndrome is not always present, and convulsions and vascular nevi may exist without glaucoma; or there may be vascular nevi and glaucoma without involvement of the brain, or convulsions may occur alone.

The vascular nevi may be found in the region of the distribution of the trigeminal nerve in the face, or they may occur in other regions of the skin. The disease is congenital and probably hereditary. Bergstrand and his associates have collected 108 cases from the literature in which there was involvement of the brain, among them, 5 cases of their own. In Bailey's book on intracranial tumors³¹ cases 19 and 20 are such cases, apparently without glaucoma.

In the brain the characteristic lesion is a calcification of small capillaries in the cortex with a multiplication and dilatation of the meningeal vessels above the cortical lesion. The dilated vessels are veins, but the walls are very thin or are thin on one side and thickened on the other side. Leiomyomatous nodules and nodular hypertrophy of the intima occur in some regions according to Bailey.

In the eye the characteristic change on which the glaucoma seems to depend is an angioma of the choroid which is of the cavernous type. A number of pathologic reports are on record. The literature is reviewed by Granström³⁵ and by Bergstrand and his co-workers.³⁶ The cavernous angioma of the choroid is not localized, but the whole choroid becomes gradually thickened from before backward and forms a disklike tumor at the posterior pole.

33. Leber,¹ p. 54.

34. Bergstrand, Olivecrona and Tönnis,²¹ p. 20.

35. Granström, K. O.: Naevus Flammeus Associated with Glaucoma, *Acta ophth.* **13**:115, 1935.

36. Bergstrand, Olivecrona and Tönnis,²¹ p. 33.

In the retina there need not be any vascular anomaly. However, in some of the reported cases vascular changes have been found there, consisting of dilatation and tortuosity of veins. Leber³⁷ mentioned the first reported case of glaucoma with vascular nevi of the skin, that of Schirmer, in which the retinal veins were dilated and tortuous, the arteries being normal. He mentioned also a case of Pantaenius, evidently with the complete Sturge-Weber syndrome, with dilatation of the retinal veins, and a case of Horrock in which there was similar involvement of the retinal veins. Mehney³⁸ reported a case in which there were markedly tortuous and anomalous veins in the retina, the arteries being normal.

Classification of Vascular Malformations and Tumors as Occurring in the Brain and in the Retina

Condition	Involvement of Brain	Involvement of Retina
Angioma cavernosum	Occurs but rarely	Probably does not occur
Angioma racemosum Telangiectasia	Occurs in all parts, especially in the pons	Occurs as the fundamental element in Coats's disease
Sturge-Weber disease	Consists of: 1. Calcification of the capillaries in the cortex 2. Dilatation of the meningeal vessels above the cortical lesion	Consists of dilatation and tortuosity of retinal veins (cavernous angioma in choroid); not in all cases
Angioma racemosum arteriale	Of rare occurrence	Occurs as dilatation and tortuosity of arteries in association with similar involvement of veins
Angioma racemosum venosum	Occurs in the meninges or in the meninges and the brain tissue below	Occurs as dilatation and tortuosity of veins alone or as part of cerebral involvement
Angioma racemosum arteriovenosum	Occurs in the brain	A rare disease in the retina; reported cases are small in number
Angioblastoma or angioreticuloma or angiomatosis of the central nervous system (Hippel-Lindau disease)	Occurs as cystic tumors of the cerebellum	Occurs as capillary angioblastomas, single or multiple, with a pair of dilated enlarged and tortuous vessels running to each tumor (Hippel's disease)
Angioglioma	Occurs as subependymal tumors in tuberous sclerosis	Occurs in extremely rare cases of microphthalmos with orbital cyst

Telangiectasia.—Telangiectasia differs from cavernous angioma in that there is some other tissue between the dilated vessels in the telangiectatic area. With the increase in dilatation of the vessels, the tissue between them is compressed and becomes atrophied. The vessels then resemble a cavernous angioma, and there is probably a gradual transition between the two forms of angioma. In the central nervous system telangiectasia occurs especially in the pons but also in other parts of the brain and spinal cord.

37. Leber,¹ p. 56.

38. Mehney, G. H.: Naevus Flammeus Associated with Glaucoma: Report of a Case, Arch. Ophth. 17:1018 (June) 1937.

In the retina telangiectasia occurs not as a part of the involvement of the brain but independently. Just as it may involve a particular vascular area of the central nervous system, so it may involve a particular vascular area of the retina. When this occurs, telangiectasia constitutes the fundamental pathologic lesion which initiates the pathologic and ophthalmoscopic changes which characterize exudative retinitis, or Coats's disease.

Angioma Cavernosum.—Cavernous angioma is a rare occurrence in the central nervous system. It does not seem to occur in the retina. The dilated blood spaces which almost resemble a cavernous angioma, such as in case 8 of Coats, are probably areas of telangiectasia in which the individual vessels have gradually become more and more dilated and have compressed the retinal elements between them.

The accompanying table gives a summary of the individual forms of angioma as they occur in the brain and in the retina.

CONCLUSION

1. Coat's disease, or exudative retinitis, is a distinct clinical and pathologic entity.

2. The fundamental pathologic element in Coats's disease is a vascular malformation involving a certain definite vascular area of the retina. This malformation corresponds to telangiectasia and involves the small vessels, the terminal units, with the formation of miliary aneurysms and dilatation of capillaries and veins with defective walls, which are subject to rupture.

3. As a result of slowing of the blood stream in the dilated vessels and of rupture of these vessels, there is a transudation of plasma into retinal tissue and the occurrence of hemorrhages there. The hemorrhages cause necrosis of retinal tissue, and the plasma and blood spread and extend externally to the retina. From the plasma, fibrin is deposited in the retinal tissue.

4. The transuded plasma and the hemorrhages incite reactive phenomena: the appearance of phagocytes derived from the cells of the layer of pigment epithelium and from histiocytes of the adventitia of vessels; the formation of fibroblasts from mesodermal elements and by metaplasia of cells from the pigment epithelium layer. The fibroblasts invade the hemorrhagic areas and cause an organization or encapsulation of these areas. When the hemorrhage is encapsulated, the central part is liquefied and contains remnants of blood elements and deposits of cholesterol.

5. Telangiectasia finds its place in a classificatory scheme of vascular malformations which involve the retina in analogy to those which involve the vessels of the brain.

PLASTIC REPAIR OF CONJUNCTIVAL DEFECTS WITH FETAL MEMBRANES

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Conjunctiva has to be replaced when a large enough area is destroyed so as to cause a high degree of symblepharon or entropion or when the socket is too small to carry a prosthesis. Most common causes of its destruction are chemical and thermic burns and trachoma. Rare causes are malignant tumor, pemphigus, tuberculous ulcer, mechanical injury and amyloid and hyaline degeneration.

Conjunctiva can be replaced primarily immediately after its loss. This has to be done after the removal of the necrotic conjunctiva. Denig¹ introduced the method by which necrotic conjunctiva is removed immediately after the chemical burn and the defect is covered with mucous membrane. However, the conjunctiva usually has to be replaced when symblepharon has already developed.

Veasey^{1a} revised the materials which are or have been used to cover the defects of the conjunctiva. These consist of grafts of conjunctiva, of the inner surface of the lips and of the conjunctiva of the rabbit and Thiersch grafts of the skin.

The ideal material for replacing conjunctiva is conjunctiva itself, but only a small piece can be taken from the same patient's other eye. For the restoration of a complete lower lid, Blaskovics² used one part of the upper fornix and adjoining tarsal conjunctiva, the corresponding strip of tarsus of the other eye being employed to build the inner layer of the lid. Some patients would refuse operation on the other normal eye in favor of the defective lid. Another material, the skin, is stiff, thick, striking in color and desquamating, and hair may grow on it. However, a sufficiently large graft is always obtainable, and therefore it is the only material for use in the restoration of the socket. The mucous membrane of the mouth is smooth and shiny and is thus a good material from the standpoint of the surface. Its

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1. Denig, R.: Early Surgical Treatment of Burns of the Conjunctiva, *Am. J. Ophth.* 3:256, 1920.

1a. Veasey, C. A., in Berens, C.: *The Eye and Its Diseases*, Philadelphia, W. B. Saunders Company, 1936.

2. von Blaskovics, L., and Kreiker, A.: *Eingriffe am Auge*, Stuttgart, Ferdinand Enke, 1938, p. 124.

disadvantage is its red color and the limit of the size available. Stellwag³ used vaginal mucous membrane on four occasions only. He abandoned the method, probably because of shrinkage of the graft. By removing the submucosa of this material, Kuhnt³ obtained less extensive shrinkage. I have used it in a case of severe progressive pemphigus in an elderly woman. The vaginal mucosa healed well, but in three months it resulted in total symblepharon just as in the other eye, on which operation was not performed. Donor and recipient belonged to different blood groups. I tried to obtain epithelization in another case by filling the orbital cavity with vernix caseosa, without any result. Kleitsman and Poska-Teiss⁴ restored vagina by this means, getting an epithelized surface after filling the artificially formed cavity several times with vernix caseosa. The authors supposed that embryonal cells of this material adherent to the wall of the cavity would produce a continuous epithelial layer. Grafts from the prepuce or labia minora have been recommended by Clay and Baird.^{4a} They used each material with good results. Grafts taken from these areas are thin, contain no hair and little subcutaneous fat and have a pinkish color. Enough tissue was available from the labia minora to restore a socket. These qualities are advantageous indeed, but many would refuse to have the graft taken from such a delicate place.

Not one of these materials is really good from every point of view in replacing the conjunctiva. An ideal material for this purpose should be a thin, smooth, transparent human tissue like the conjunctiva; it should be sterile and obtainable in sufficiently large amount.

Fetal membranes seem to have all these requirements; in addition, they are elastic and carry but few vessels, and the patient does not have to undergo another operation. They have been used by Burger⁵ to fit the artificially formed vagina. I obtained such membranes by cesarean section from patients at the Second Women's Clinic, University of Budapest, who had a negative Wassermann reaction. The membranes were kept in tepid Locke solution. One to fifteen hours elapsed between the cesarean section and the implantation. I used this material in 6 cases of symblepharon (8 operations) and in 2 cases in which the socket

3. Cited by Elschmig, A.: *Augenärztliche Operationslehre*, Berlin, Julius Springer, 1922, vol. 1, p. 410.

4. Kleitsman, R., and Poska-Teiss, L.: *Ueber die Anwendung von Vernix caseosa bei der Bildung einer künstlichen Scheide und histologische Untersuchung der letzteren*, *Zentralbl. f. Gynäk.* 59:755, 1935.

4a. Clay, G. E., and Baird, J. M.: *Restoration of the Orbit and Repair of Conjunctival Defects with Graft from the Prepuce and Labia Minora*, *J. A. M. A.* 107:1122 (Oct. 3) 1936.

5. Burger, K.: *Experimental and Clinical Studies on Transplantation of the Fetal Membranes*, *Orvosi hetil.* 82:800, 1938.

had to be enlarged. Symblepharon was caused in 3 cases by lime burn and in 1 case each by phenol, calcium carbide and pemphigus.

The technic of the operation was the same as proposed by me in 1929.⁶ The conjunctiva is undermined from the limbus to the margin of the lid, even if there is but a small portion of fornix left. The undermined conjunctiva is fixed to the lid with two to three mattress sutures to build the palpebral conjunctiva. If there is a complete symblepharon of one lid, I try to line the lower lid with a pedunculated flap of the upper bulbar conjunctiva. While in previous operations I applied mucous membrane of the mouth to cover the defect of the fornix and bulbar conjunctiva produced by the previous procedure, in these 10 operations fetal membranes were used. The graft is fixed to the tendon of one or more rectus muscles, which gives a fixed point on



Result of conjunctival plastic repair with fetal membranes. The patient, a woman aged 28, had had the left eye injured with phenol on May 12, 1938. She was admitted to the State Eye Hospital on July 13. There was no left upper fornix, and the upper edge of the tarsal conjunctiva bent over the limbus. Transplantation of fetal membranes was done on July 21. This picture shows the condition on September 9. The vessels of the bulbar conjunctiva continue in the transplant.

a firm base and avoids shrinkage. The graft covers the fornix, and its edge will unite with that of the conjunctiva lining the lid. The graft is not allowed to make folds but just covers the sclera and fornix smoothly.

When fetal membranes are used the chorion surface is placed on the wound following removal of tense fibers of connective tissue, if any are present. The amnion forms the free surface. In 1 case of total symblepharon of both the upper and the lower lid the fetal membranes were not sutured to the rectus muscles, but a contact glass was covered

6. de Røth, A.: Bindehautplastik, in *Concilium ophthalmologicum*, s'Gravenhage (The Hague), Netherlands, 1929.

with the membranes and placed on the eye. The glass was removed on the seventh day when the membranes adhered to the sclera and the lid. In all cases a binocular dressing was applied for three days when the bandage was first changed.

The fetal membranes took in every case, and for three weeks they showed no change. In the next weeks the graft shrunk, and ten weeks after operation it seemed to have disappeared, but there was always more fornix left than before operation. Thus the shrinkage was rather extensive, but the membranes left seem to transform to conjunctiva. However, 1 patient with symblepharon of the upper lid due to phenol had a good result but with a little shrinkage. Three months after operation there was a good upper fornix, as shown in the accompanying illustration. The new vessels of the graft show a course similar to that of the normal conjunctival vessels. One never sees such newly formed vessels in the transplanted mucous membrane. In a case of lime burn the upper fornix had to be restored. The fetal membranes covered the bare upper half of the cornea as well. Ten weeks after operation there was a shallow fornix and the cornea was shiny. In the case of pemphigus the restored lower fornix was shallow two months after the operation. In 3 other cases of symblepharon the late result was poor with nearly total shrinkage of the membrane.

Thus the results were poor or insufficient in but 1 case. The method deserves further study for the following reasons:

1. The embryonal tissue used has a property of being transformed to conjunctiva. The epithelium of the membranes excised two and four months after transplantation showed the same histologic structure as the epithelium of the bulbar conjunctiva.

2. In the successful case the course of the new vessels in the graft was like that of a normal conjunctiva. In no other form of graft may this be observed.

3. The fetal membranes covering the cornea resulted in a shiny transparent tissue. Whether it was the graft itself or whether the graft was replaced by normal corneal epithelium, I do not know.

At present I should confine the indication of using fetal membranes in conjunctival plastic operations to cases of symblepharon when for any reason mucous membrane of the mouth is not obtainable; i. e., when there is disease of the mouth, when the patient refuses operation in the mouth and, further, when the symblepharon includes the cornea as well, for mucous membrane of the mouth gives a poor cosmetic effect when covering the cornea.

SANTA LUCIA, PATRONESS OF THE EYES

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Ophthalmologists who practice in sections where there are many Italians frequently hear reference to Santa Lucia as a patron saint of those who suffer from ocular troubles. Having been presented with a statuette of the saint by a grateful patient, I became interested to learn more of her life and the reasons why she is supposed to make special intercession for those afflicted with disorders of the eyes. It soon became evident that there are both a historical account of Saint Lucy and a legend which sprang up much later, the former entitling her to a place as a virgin martyr and the latter making her a special saint to those with ocular complaints.

Certain facts of the life of Saint Lucy appear to be well authenticated, and she is one of the few women whose names appear in the canon of the mass of Gregory the Great. Born of Christian parents in Syracuse, in southern Italy, at the end of the third century (283), the young girl early evinced profound religious zeal and dedicated her virginity to Christ. Her father having died when Lucy was an infant, she was brought up by her devoted mother, Eutychia. The latter suffered for several years from a flux of blood, which none of the physicians was able to cure. Finally Lucy persuaded her mother to make a pilgrimage to Catania, to the tomb of Saint Agatha, where many miraculous cures were reported to have occurred. While Eutychia was praying at the tomb the hemorrhage ceased, and mother and daughter returned to their home city. Apparently sensing that this was a favorable time to make a request, Lucy persuaded her mother to give her her inheritance, that she might distribute it to the poor. Against her wishes, the beautiful girl had been betrothed to a noble, but pagan, youth of Syracuse, and when the latter learned what she had done with her riches he denounced her to Paschasius, the governor, as a Christian. This occurred during the early part of the fourth century (303), during the reign of Diocletian, when the persecution of the Christians was at its height.

There are numerous accounts of the trial of Lucy before Paschasius, some even recounting verbatim the charges against her, and her replies. In general, it seems believable that she was persecuted as a Christian and that she was condemned to a life of shame. When Paschasius ordered Lucy to be taken away, the crowd of attendants

began to push and jostle her, but she stood immovable. Exasperated, the tormentor commanded that she be carried away by force, but the soldiers were unable to stir her. A yoke of oxen was then brought in, but they also were powerless to move the girl. Certain accounts relate that a fire was built about the victim and that the roaring flames had no more effect than in the case of the three men thrown into the fiery furnace. As a last resort, a sword was drawn and her throat pierced, causing her to bleed to death. As Baring-Gould puts it, "with saints as with witches, when everything else proves ineffectual to hurt them, cold steel breaks the charm."

The account of the life and death of Santa Lucia to this point is generally accepted by church historians, but no mention had been made of any fact that would entitle her to be considered as a protectress of those with afflictions of the eyes. So far as is known there is no early written account of this association, but during the Middle Ages and from then on painters and sculptors have frequently represented Santa Lucia with her eyes on a salver or at times spitted on a sharp instrument. In the Catholic Encyclopedia¹ it is stated "The story of Santa Lucia is to be traced back to the Acta, and these probably belong to the fifth century. Though they cannot be regarded as accurate, there can be no doubt of the veneration that was shown Santa Lucia by the early Church." This source makes no mention of the eyes whatsoever. The first known writer to give an account of the life and passion of Santa Lucia from the Acta was the English bishop, St. Aldhelm of Sherborne, at the end of the seventh century. Since, however, the Acta are admitted to be an untrustworthy source of information, the factual value of the bishop's literary efforts seem slight.

As Mrs. A. B. Jameson stated in "Sacred and Legendary Art,"² there is no mention in the Acta or in any of the earliest legends of the loss of the eyes. Some of the early painters used the device of placing an emblem of an eye, or eyes, near to her to express her name, Lucia (from the Latin *lux*). From this source seems to have come the invention of the additional incident in her story—"a signal instance of the convergence of the image or metaphor into a fact, which I have so often had occasion to notice."

In the literature there are many references to the story which developed, apparently several hundred years after the historically recorded martyrdom of Santa Lucia, of the manner in which she lost her eyes. The story relates that a youth of her city became enamored

1. St. Lucy, in The Catholic Encyclopedia, New York, The Encyclopedia Press, Inc., 1913, vol. 9, p. 414.

2. Jameson, A. B.: Sacred and Legendary Art, Boston, Houghton Mifflin Company, 1865, vol. 2.

of her and by messages, gifts and all methods known to ardent lovers attempted to win her favor. In his letters and pleadings the young man protested that it was the beauty of her eyes which inflamed him, permitting him no rest by day or night. Fearing that such ardor would be destructive to the youth and that she herself might prove faithless to her vow of chastity, Lucy dwelt on their plight. Recalling the words of Christ, "If thine eye offend thee, pluck it out and cast it from thee," the future saint determined to follow this advice literally and so plucked out her eyes and sent them to her lover on a salver, with the message, "Here thou hast what thou hast so much desired. and for the rest, I beseech thee, leave me now in peace." The story proceeds to a worthy conclusion, for the young man, utterly astonished and torn by remorse and grief, became a convert to Christ and lived ever afterward a life of virtue and chastity.

"But God would not suffer that the blessed Lucia, having given proof of her courage and her piety, should remain blind, for one day, as she knelt in prayer, behold!—her eyes were restored to her more beautiful than before. And if anyone doubts of the great miracle let him consult the writings of that learned and praiseworthy man Fillipo Bergomene, and also of the famous Spaniard Don Juan Moldonato, where they will find it all set down as I have related. And this is the reason that Santa Lucia is invoked against blindness and all diseases of the eyes, and that in her effigy she is represented bearing two eyes on a dish."² Here lies the explanation for the curious fact of the martyr sacrificing her eyes by autoenucleation and yet having two other normal and beautiful eyes.

Another legend relates that a Roman emperor fell in love with Lucy's beautiful eyes and that when she rejected his suit he caused her eyes to be destroyed. This account is later than the one previously cited and seems to carry no weight.

According to Alban Butler,³ Santa Lucia was honored at Rome in the sixth century among the most illustrious of the virgin martyrs. Her festival was kept in England until the Reformation as a festival of the second rank, during which no work was done except tillage. In "Weather Folk-Lore"⁴ is the statement, "Lucy light, the shortest day and the longest night" (December 13, the day of her martyrdom). Santa Lucia is patroness of Syracuse, of Mantua, of the laboring poor, of tillers of the ground and of sight and the eyes; she is also thought to furnish protection against dysentery and hemorrhage of all kinds.⁵

3. Butler, A.: *Lives of the Saints*, revised by Herbert Thurston, New York, P. J. Kenedy & Sons, 1926.

4. Swainson, C.: *Weather Folk-Lore*, London, Trübner & Co., 1885.

5. Dunbar, A. B. C.: *Dictionary of Saintly Women*, London, George Bell & Sons, 1904, vol. 1, p. 469.

Saint Lucy, worshipped from early times, has been the inspiration of countless artists. In the earliest paintings and sculpture she is shown holding a palm (fig. 1), the symbol of her death for Christianity's sake. In a church in Cajazzo, Italy, there is a twelfth century fresco showing Saint Lucy carrying a lamp, symbolic of illuminating grace. But more often, especially as in later years, she was depicted holding a dish with two eyes on it (fig. 2). More rarely she holds a rod or a dagger which pierces the eyes (fig. 3).



Fig. 1.—Saint Lucy, by Della Robbia. This work, sixteenth century, is to be found in the church of Santa Maria a Ripa, at Empoli, Italy. (Photograph by Alinari.) This and the following illustrations are taken from a small and obviously old book entitled "*L'art et les saints—Sainte Lucie*," by Georges Goyau, Paris; Henri Laurens, éditeur, 6, Rue de Tournon. On one page, at the bottom, are the words: "Evreux, Imprimerie ch. Hérissé (11-21). No date is given.

There is a manuscript, written in 1220, preserved in the Dome of the Milan Cathedral and signed by Gottofredo of Bussero, stating that Lucy took her name from "light." Cardinal Frederick Borromeo, in the sixteenth century, in his book "*On Sacred Paintings*" mentioned

a connection between Saint Lucy and the pagan divinity Lucini, who was also recommended for ocular troubles.

Not only have painters and sculptors done homage to Santa Lucia, but great poets and writers have added to the luster of her name. It is stated that Dante wept so much over Beatrice that his eyes were affected and that Saint Lucy, for whom he professed great devotion, cured him. In the "Convivio" he stated that if he had to name two



Fig. 2.—Saint Lucy, by Giovanni Spangna. This painting, sixteenth century, is to be found in the galerie du capitol, at Rome, Italy. (Photograph by Alinari.)

cities he would call one Mary and the other Lucy. In the "Divine Comedy," Dante is represented as distracted by the memory of Beatrice, and from Heaven a "gentle lady," the Virgin Mary in person, saw with pity the soul of the poet so distraught with the death of his love. On Lucy's feast day, December 13, he believed that he heard Mary say to Lucy, "His faith needs your help; I recommend him to you." Whereupon Lucy led Dante and Virgil in the search for Beatrice, and as they traveled over the unfamiliar land they were kept constantly in sight by Lucy. As he fell asleep from fatigue, Lucy appeared to Virgil and

offered to carry Dante on his voyage. In this manner they reached the gateway to Purgatory, when Lucy disappeared. Finally, in Paradise, she reunited Dante and Beatrice. Dante placed Lucy in one of the highest seats in Paradise, in front of Adam, the head of all humanity, and close to the throne of the highest.

Christopher Columbus is related to have been cured of ocular trouble by this patron saint and to have named one of the islands of the



Fig. 3.—Saint Lucy, by Bernardino Luini. This painting, sixteenth century, may be found in the Monasterio Maggiore at Milan, Italy.

Lesser Antilles, discovered on December 13, Santa Lucia, in her honor. Likewise, Alessandro Monzoni called one of his principal characters Lucia in his greatest work, "I promessi sposi."

Throughout the Middle Ages, especially in Germany, the night of Saint Lucy, December 13, was thought to be the time when phantoms and demons roamed the earth, spreading ills. Saint Lucy was supposed to be about that night warding off the evil spirits and their malign effects. Even today her feast is observed regularly in Sweden, for on



Fig. 4.—Saint Lucy, by Carlo Dolci. This painting, seventeenth century, hangs in the galerie des offices, at Florence, Italy. (Photograph by Alinari.)



Fig. 5.—Saint Lucy. An engraving from "De claris mulieribus" by J-P. de Bergame.

the dawn of her festival day a girl of each family, dressed in white and wearing a lighted crown, offers coffee and *Lusia* cakes in honor of the saint, who symbolizes the returning daylight. The most beautiful "Lucia" is selected to lead a triumphal procession in the capital.

The symbolism of a word suggesting definite characteristics is shown in several similar instances. In France Saint Claire is invoked for ocular diseases, because *claire* means clear. In Germany Saint Augustine was supposed to intercede for thoses so afflicted because of his name (suggesting *Auge*, eye).

In the realm of art the earliest paintings of Saint Lucy refer to her trial and martyrdom. It is only later that the symbolism of light and the eyes appears. A painting by Carlo Dolci shows her with rays of light diverging from the wound in her neck (fig. 4), from which she died. Della Robbia represented her with a palm leaf in one hand and a book in the other. The earliest depiction of Saint Lucy known in which there is presented the feature of the loss of the eyes is an engraving by de J.-P. de Bergame, of Ferrara, Italy, in 1497 (fig. 5). A fifteenth century painting by Filippino Lippi, in the Cathedral of Prato, Italy, shows her carrying a lamp in her hand. However, in the sixteenth century the artists almost always pictured her with her eyes on a saucer, and it would therefore seem to be about this period that the legend of the loss and restoration of the eyes became popularly known. A painting by Bernardino Luini in Milan, Italy, shows the eyes held on an awl or similar instrument—a representation not in the best of taste, judged by modern standards.

Among the great painters who have depicted Santa Lucia are: Della Robbia, Antonelle Gaginia, Antonio Riccio, Parmegianino, Bernardino Luini, le Perugin, Lello da Velletri, Carlo Dolci and Giov de Spagna.

Whether or not one believes in the power of saintly martyrs to intercede with the Almighty in the cure of afflictions, there can be no doubt but that much mental comfort and support come to those who feel the need of mediators. And among these saints stands Santa Lucia, who intercedes for those who suffer one of the greatest known afflictions, the loss of sight.

BILATERAL UVEITIS ASSOCIATED WITH DETACHMENT OF THE RETINA (HARADA'S DISEASE)

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The epochal discoveries of Gonin regarding the pathogenesis and surgical treatment of noninflammatory detachment of the retina and the work of various authors in the ensuing years enriched and fundamentally changed the knowledge of this subject. The differentiation between inflammatory and noninflammatory detachment became more distinct. Detachments of the latter type, exhibiting changes in the vitreous and the peripheral part of the retina, occur mainly in persons with myopia and in the senium. The former type, in which the separation of the retina is caused solely by the inflammatory exudation or transudation, is distinguished by the absence of holes or tears. Clinically, in cases of inflammatory detachment one cystlike prominence is usually seen in the lower part of the retina. There is no floating movement present; the detachment, rather, reminds one of a balloon with highly stretched walls. There is a sharp distinction between the two types as to prognosis and treatment, the detachment following or accompanying inflammatory changes in the other membranes of the eye necessitating only symptomatic treatment but no surgical intervention.

Inflammatory changes of a sclerotic nature are known to cause detachment of the retina. Purtscher¹ first observed a case of scleritis complicated by separation of the retina with spontaneous healing. A similar case was described by Kamocki.² The condition in each case was unilateral; in Purtscher's case the patient suffered from iritis also. The separation of the retina took place in the area corresponding to the scleral pathologic process and, according to the explanation of Purtscher, was due to the disturbance of the lymph circulation following the infiltration of the sclera. The case of Pichler belongs to the same group. The patient, a man 53 years of age, suffered from recurring scleritis. In the course of one of the serious attacks accompanied by symptoms of periostitis of the adjacent orbital wall, a detachment of the retina established itself, disappearing within seven months. There was no iritis present. The attacks of scleritis in the other eye were not

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1. Purtscher: *Centralbl. f. prakt. Augenh.* **15**:334, 1891.

2. Kamocki, V.: *Centralbl. f. prakt. Augenh.* **16**:15, 1892.

combined with retinal detachment. Leber,³ in his excellent work on retinal disease, mentioned retinal separation in the course of scleritis and episcleritis and tried to explain the mechanism as being similar to that in cases of phlegmon of the orbit, in which the infiltration proceeds along the emissaria to the inner membranes of the eye. The newer literature on the subject contains reports of cases of Horay⁴ and Csillag⁵ which possibly belong in this group.

Csillag observed a woman 34 years of age with marked scleritis in the upper part of the sclera, ciliary congestion, absence of changes in the anterior segment, a hyperemic disk and detachment of the retina in the upper part of the nasal quadrant without tears or holes. The detachment of the retina progressed, and in the course of the disease the lower and temporal parts were separated too, accompanied by exophthalmos, chemosis of the conjunctiva and serous exudation in the retina, enveloping the vessels. The condition remained unilateral and resulted in complete reattachment of the retina without operative intervention. Doubtlessly the primary disease, the circumscribed scleritis, was instrumental in producing the detachment in the corresponding area of the retina. With the progress of the inflammatory changes, the detachment occupied the lower and temporal parts of the retina as well. The explanation given was that the acute inflammatory reaction caused edema of the choroid, the stasis of tissue fluid sank downward due to the loose consistency of the choroidal structure and the increased volume resulted in detachment of the retina. Further proof of the mechanism of edema is given by the fact that after absorption of the fluid and disappearance of the edema, the fundus did not reveal any pathologic changes referable to previous choroidal inflammation.

The chapter dealing with retinal detachments in the course of deep scleritis was further enriched by a contribution of Karasek,⁶ in whose case posterior scleritis led to neuritis, then retrobulbar neuritis, paresis of the abducens nerve and a rather flat retinal detachment of the macular region (about +3.0 diopters), which is explained by exudation between the sclera and the choroid with an ensuing inflammatory involvement of the choroid and retina due to the infectious toxic substances.

Meisner⁷ contributed 4 cases of his own to this review, emphasizing the rarity of cases and the favorable prognosis. Among 150 cases of detachment, he observed only 4 of the inflammatory type in which

3. Leber, in von Graefe, A., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, Wilhelm Engelmann, 1915.

4. Horay, G.: *Klin. Monatsbl. f. Augenh.* 95:656, 1935.

5. Csillag, F.: *Klin. Monatsbl. f. Augenh.* 98:206, 1937.

6. Karasek, O.: *Klin. Monatsbl. f. Augenh.* 95:645, 1935.

7. Meisner, W.: *Schweiz. med. Wchnschr.* 67:788, 1937.

surgical intervention was not indicated and the symptomatic treatment resulted in complete healing. In his first case, that of an emmetropic woman 40 years of age, there was a detachment of four weeks' duration occupying the lower temporal part of the retina. No holes or folds were present, and there were a few small old choroidal patches at the margin of the detachment. The cutaneous test for tuberculosis was positive. After reattachment, visible changes of the choroid in the macular area took place. The case is classified as one of tuberculous choroiditis with retinal detachment.

The patient in the second case gave a history of painful scleritis of many months' duration before a large detachment developed in the lower part of the retina. There were no ruptures and no inflammation in the anterior part of the eye. Histologic examination of the enucleated eye revealed the presence of a tumor of the sclera of fibromatous appearance, small foci of calcification, infiltration suggesting a tuberculous origin and similar changes in the posterior part of the sclera and choroid.

The patient in the third case, a woman 30 years of age, showed marked ciliary congestion of the right eye, increased corpuscular elements in the aqueous, numerous delicate deposits on the anterior capsule, fibrinous masses at the dilated pupillary margin, opacities in the vitreous, a hazy and swollen disk and detachment in the lower part of the retina without the formation of folds. The left eye, simultaneously, was affected by similar, but less marked, inflammatory changes in the anterior segment without retinal separation. As the disease advanced, fibrinous nodules appeared in the left iris and a detachment of the retina in the lower temporal part resulted. After healing, depigmentation and numerous smaller and larger pigment patches at the site of the previous detachment were evident. Serologic and tuberculin tests were negative.

The patient in the fourth case, a man 54 years of age, suffered from rheumatism and recurring iridocyclitis in both eyes. The right eye showed ciliary congestion, pigmentation of the anterior capsule, a normal iris, a clear vitreous and an extensive detachment of the entire lower part of the retina without tears. The other eye was normal, only remnants of previous iritis being discernible.

All 4 patients had in common retinal detachment as a sequel to inflammatory changes in other membranes of the eyes. With the exception of the third patient, all showed unilateral involvement. The first presented tuberculous choroiditis; the second, severe tuberculous involvement of the sclera and choroid, and the third and fourth, inflammatory changes of the uvea, the iris and the ciliary body. The detachment of the retina developed in the course of acute inflammatory

reaction and healed under symptomatic treatment without the necessity of surgical aid. The resorption of the exudation resulted in complete reattachment.

The unilateral, purely inflammatory retinal detachment forms the bridge to the other type of detachments in which the separation is simultaneously bilateral and is the main feature of the symptom complex. The detachment is so much the important aspect of the pathologic process that the other possible inflammatory symptoms are apparently of lesser significance. Harada⁸ in 1926 first described bilateral detachment in the course of low grade uveitis as an independent disease entity. The first publication came from Japanese sources only (12 cases). It seems that Salus⁹ was the first European to publish an observation belonging to this group. Harada's disease presents an acute, diffuse, exudative choroiditis or uveitis bordering on diffuse uveitis accompanied by deafness, poliosis and loss of hair and in turn on sympathetic uveitis. However, the typical onset, the extensive retinal detachment and the relatively favorable prognosis of the spontaneous reattachment are features warranting the consideration of the separation as an entity. Harada's disease is invariably bilateral; the onset is a rather acute one accompanied by headaches and occasional vomiting or nausea and characterized by either lack of, or low grade, inflammatory symptoms in the anterior segment of the uvea. In the early stage, opacities of the vitreous, edema of the disk, suggestive of papillitis, and yellowish discoloration of the retina are present; these are followed after a few weeks' duration by extensive detachment. The detachment has a definite tendency to disappear gradually within a few weeks, and the healing results in unexpected good visual acuity, in view of the previous extensive and severe pathologic changes. The eyeground picture is that of depigmentation, the redness becoming lighter with the presence of patches of pigment in the retina due to proliferation of pigment elements.

A patient of Salus, a woman 38 years of age, had headaches at the onset; the eyes were white, but numerous delicate grayish white deposits were present, especially in the right eye. The irides exhibited small nodules or vesicles similar to those seen in cases of tuberculosis of the iris. Delicate opacities of the vitreous, markedly swollen disks and enormously dilated and tortuous retinal veins were present. Yellowish gray cloudiness was observed in the retina. In the course of the disease posterior synechiae appeared, the nodules of the irides disappeared and the retinal infiltration became more extensive. In the seventh week of the disease, simultaneous detachment of the retina occurred in each eye, progressing from the periphery toward, and finally reaching, the

8. Harada, cited by Salus.⁹

9. Salus, R.: *Klin. Monatsbl. f. Augenh.* 89:84, 1932.

disk. The detachment disappeared about the same time in each eye simultaneously with the other inflammatory symptoms (scleral puncture was performed on the left eye). After the disappearance of the latter, numerous posterior synechiae with a delicate occlusion membrane were present. The fundi appeared albinotic due to the depigmentation, with visible choroidal vessels not exhibiting any vascular pathologic process, but a few proliferative patches of pigment were noted. In the periphery there were a few small white patches suggestive of sympathetic choroiditis and at the posterior pole wide stripes of organized exudate with delicate pigmentation. The case belongs, according to Salus, to the group of cases of Harada's disease, notwithstanding the few apparent discrepancies, such as pathologic involvement of the anterior segments marked by the presence of deposits on the posterior surface of each cornea and nodules in the irides, which he did not consider tuberculous, the intracutaneous tuberculin test showing only a slight reaction.

Salus' case appears more remarkable because of the interesting fact that a sister of the patient two months later suffered from similar iridocyclitis with formation of nodules in the irides. The disease appeared simultaneously in each eye and involved almost exclusively the anterior parts only, the choroid remaining free of changes with the exception of a few small peripheral patches.

Horay's patient, a man 46 years of age, had in the left eye opacities in the vitreous, detachment of the retina in the lower part without tears and chorioretinitic patches in the periphery; the detachment flattened out ten days after scleral coagulation. A short time later a scleritic nodule developed in the normal right eye; later there developed chemosis of the conjunctiva, periosteal irritation at the temporal part of the orbit and detachment of the retina corresponding in area to that of the scleral infiltration. Later, there were visible exophthalmos and total detachment of the retina. The detachment disappeared without operative intervention. Reexamination after three months showed a picture of hazy margins of the disk, dilated retinal veins and a yellowish red retina with patches of pigment; in the region of the ora serrata, a flat, ring-shaped, almost imperceptible detachment remained.

The extreme rarity of similar observations and the somewhat meager reports in the literature have prompted the publication of 2 cases which, doubtlessly, belong in the group of cases under discussion and demonstrate that even within the same group the prognosis and ultimate outcome of the given case may show extensive variations.

REPORT OF CASES

CASE 1.—A man 22 years of age, of Italian parentage, was first seen on Oct. 30, 1937. He complained of progressive loss of vision for the preceding six weeks. He had had "grip" seven weeks prior to the onset of blurred vision. About three

days after his recovery from "grip" he noticed loss of vision on the nasal side of his left eye. The vision became progressively worse in both eyes, so that at the end of four weeks he retained only perception of light and was unable to verify objects.

He could not recall having had any diseases of childhood. There was no history of cardiac, respiratory or metabolic disease or of syphilis or gonorrhea. The family history did not reveal any ocular disease. There was no history of tuberculosis. He had fractured his right arm many years ago.

Physical examination showed a well developed white man. The tongue was moist, and the pharynx was not injected. There were a few small movable nodes in the neck and no enlargement of the thyroid. The heart, lungs and abdomen were normal. The reflexes were physiologic.

The right palpebral fissure was smaller than the left. Each eye showed conjunctival and slight ciliary congestion. The endothelial layer showed marked edema. The lower part of the cornea had a tissue paper-like appearance with a few small round grayish deposits. The irides were somewhat swollen, and there were no newly formed capillaries. The pupils dilated, but not to the maximum (atropine); the diameter of each pupil was about 5 mm. On the anterior capsules there was delicate pigment dust, and on the area corresponding to the undilated pupillary margin, larger patches of pigment. The vitreous in each eye was very hazy, the haziness being of a diffuse character. There were no large floaters, only a dustlike variety. There was an enormously extensive retinal detachment with a maximum prominence of +16.0 diopters in the right eye, involving the nasal, lower and temporal parts of the retina, and in the left eye involving the lower and nasal parts of the retina.

Roentgen examinations were made (Dr. J. Furst). The accessory nasal sinuses showed insufficient evidence of gross pathologic change to be of diagnostic value. There was, perhaps, a slight degree of congestion of the left ethmoid sinus. Exposures of the thorax showed no evidence of active parenchymal infiltration or pleural involvement.

The urine was clear and acid and had a specific gravity of 1.202; there was no albumin or dextrose. The blood count showed: 12,200 leukocytes, 73 per cent polymorphonuclears, 12 per cent lymphocytes, 8 per cent endothelial leukocytes, 4 per cent eosinophils, 1 per cent basophils, 68 per cent hemoglobin, and 3,850,000 erythrocytes. The urea nitrogen content was 132 mg.; the uric acid, 32 mg.; the creatinine, 1.3 mg., and the sugar, 88 mg. per hundred cubic centimeters. The Wassermann and Kline reactions were negative. The sedimentation rate of the blood was 50 mm. the first hour and 80 mm. the second hour. Cultures from the right and left nostril showed *Staphylococcus albus*; from the throat, *Streptococcus haemolyticus* and *Micrococcus catarrhalis*. A filtrate (0.2 cc.) of *Str. haemolyticus* given intradermally caused no reaction. The Mantoux test was negative.

Within the next ten days, the ciliary congestion cleared up completely. Both eyes became extremely hypotonic. The irides in the peripheral parts were almost touching the posterior corneal surfaces due to multiple circumscribed bulging areas; the pupillary part of each iris was drawn backward toward the vitreous. The backward pull was not funnel shaped but almost at an angle of 90 degrees, especially in the nasal part of the right eye.

Within the ensuing seven days numerous large deposits appeared all over the posterior corneal surface of the right eye. Only a few small deposits were seen in the lower part of the left eye. The pupillary margin in each eye was circularly attached to the lens with a few delicate capillaries growing from the iris into the thin seclusion membrane, covering the entire pupillary area. The anterior chambers

were extremely deep, the lens-iris diaphragms being drawn back toward the vitreous. A few grayish nodules were seen in the irides. The fundi were invisible. Hypotonia of extreme degree persisted.

Within the next four weeks vision in each eye improved to counting of fingers at 1 meter. The right disk was hazy and the margins veiled. In the nasal part there was delicate pigmentation and in the lower part, numerous round white pinhead-sized patches arranged in grapelike clusters. There were a few large opacities in the vitreous of the left eye and delicate pigmentation in the retina. The previously extensive detachment of the retina completely disappeared.

Two months later the right eye showed slight ciliary congestion but a markedly increased number of deposits; the entire posterior surface was covered, carpet-like, with large round yellowish deposits. The left eye did not show any ciliary congestion but more deposits. The vitreous in each eye was diffusely hazy, but the fundi were clearly visible. The fundi were yellowish and had less vivid redness than usual. Complete reattachment of the retinas occurred. The disks were somewhat veiled, and the veins were dilated and engorged. Over the retinas there were small irregularly outlined patches of brownish pigment, occasionally covering the retinal vessels; their relation to the retinal vessels revealed the different localization in the various layers of the retinas. In some parts of the retinas there were pinpoint to pinhead-sized white patches with delicate, rather black pigmentation similar to the changes occurring in cases of healed sympathetic choroiditis. The vision in the right eye was 20/200 and in the left eye 20/50; the patient did not accept glasses. The tension (Schiötz) in the right eye was 10 mm. of mercury and in the left eye 12 mm.

The patient was seen again two months later. The inflammatory symptoms in the anterior segments had not increased, but the fundi were invisible because of rapidly developing complicated cataracts. Hypotonia persisted; the vascularization of the seclusion membrane was more marked than before.

General treatment consisted of injections of foreign protein intramuscularly, followed by mercury inunctions and tuberculin.

CASE 2.—A girl 18 years of age, of Italian extraction, noticed a sudden diminution of vision three weeks before examination. There was pain in each eye at the same time, and the eyeballs turned very red. General examination gave negative results.

The urine was clear and showed an acid reaction, a specific gravity of 1.023, 2 mg. of albumin, no dextrose or casts and 15 leukocytes (high power field) and 2 epithelial cells. The blood count revealed: 8,700 leukocytes, 70 per cent polymorphonuclears, 19 per cent lymphocytes, 5 per cent endothelial leukocytes, 1 per cent basophils, 3 per cent myelocytes (stab forms), 81 per cent hemoglobin and 4,200,000 erythrocytes. The urea nitrogen content was 20 mg. and the blood sugar 100 mg. per hundred cubic centimeters. The Wassermann and Kline reactions were negative. The sputum was negative.

Roentgen examinations were made (Dr. J. Furst). The accessory nasal sinuses showed fairly good aeration and ventilation. An exposure of the thorax showed insufficient evidence of gross pathologic changes to be of diagnostic value.

The reaction to the Mantoux test (with a 1:1,000 dilution of tuberculin) was 3 plus. (After twenty-four hours there was redness and induration 0.5 cm. in diameter.)

Vision in the right eye was 15/200 and in the left 15/100; there was no improvement with glasses. Marked conjunctival and ciliary congestion was present in each eye. The slit lamp showed the posterior surface of each cornea to be tissue

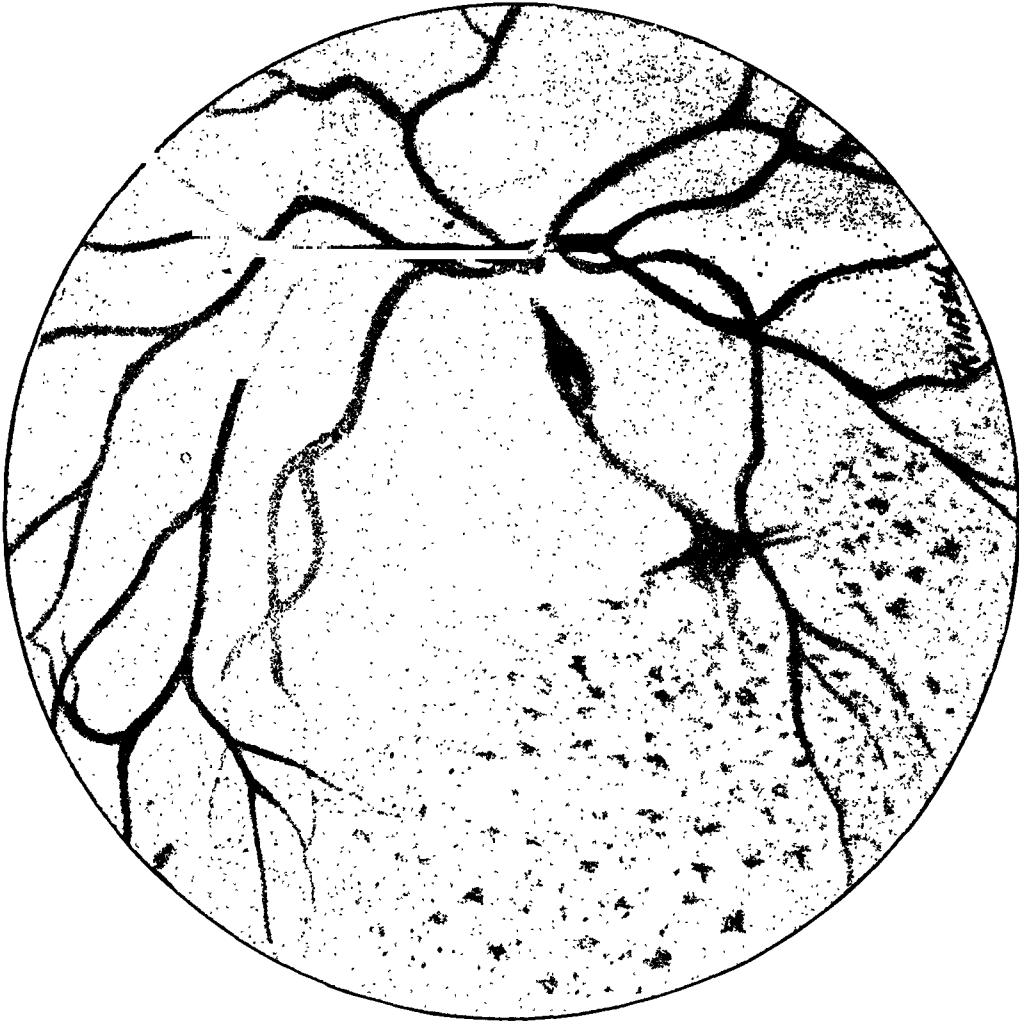


Fig. 2.—Left eyeground of the same patient.

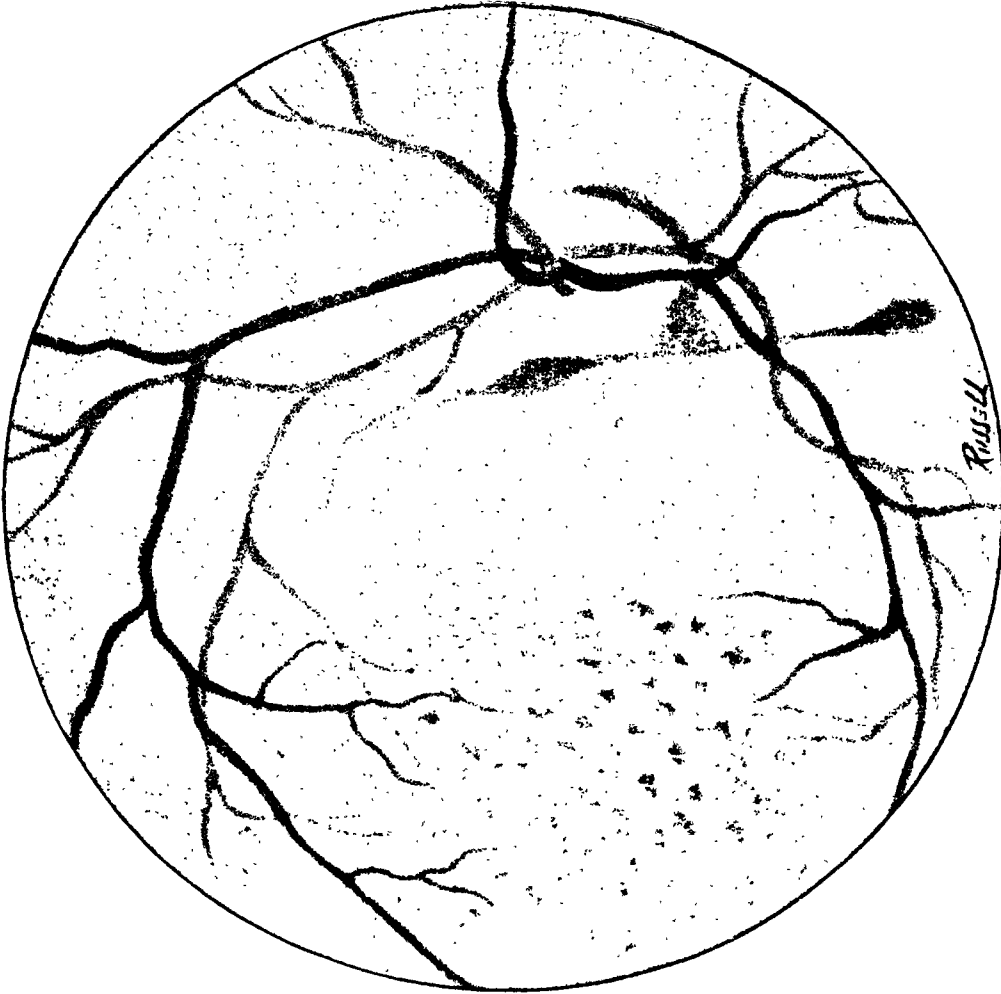


Fig. 1 (case 2).—Right eyeground. This illustration and also figure 2 were made about three months after reattachment of the retina took place. It is interesting to note the yellowish hue due to depigmentation. The small pigment patches are of a brownish color and aggregate in the macular region. Since the drawing of the pictures, the patches have increased somewhat in number but not in size. On both eyegrounds a stripe-shaped pigmented line, somewhat elevated, is clearly visible.

paper-like and covered with delicate fibrinous stripes. The anterior chamber of the right eye contained fibrinous exudation. There were numerous hyperemic posterior synechiae in the irides, especially in the upper part of the right iris. No deposits were noted. A delicate fibrinous membrane was present in both pupillary areas. There was an extensive retinal detachment in the lower part of each retina; the maximum prominence in the right retina was $+ 8.0$ diopters and in the left, $+ 12.0$ diopters. The detachment was more extensive in the left eye than in the right. The vitreous in each eye was diffusely hazy, and the retinal veins were markedly dilated and engorged.

Nine days after the initial examination there were a few deposits on the right eye and more deposits in the lower part of the cornea of the left eye. The deposits were white and star shaped. The pupils dilated to the maximum. There were almost circular posterior synechiae and increased circulation and pigmentation of the anterior capsules. The anterior chambers were deep and the irides markedly retracted. The tension (Schiötz) was 12 mm. of mercury in each eye.

Fifteen days later the deposits were markedly increased in number and size. In the right eye, temporal from the disk, there was one large stripe-shaped area of retinal hemorrhage. Complete reattachment of the detached part of the retina had occurred in each eye.

Three months later both eyes were white. There were no symptoms of irritation or synechiae, and the vitreous was clear. Vision in the right eye was 15/30; with a $+ 0.5$ sph. $\ominus + 0.5$ cyl., axis 180 it was 15/15. Vision in the left eye was 15/30; with a $+ 0.25$ sph. $\ominus + 0.5$ cyl., axis 165 it was 15/20. The tension (Schiötz) was 22 mm. of mercury in each eye.

Figures 1 and 2 show the condition observed in the eyegrounds. The fundi were definitely pale, somewhat yellowish in comparison with the usual red color. On the fundi, especially in the macular region of each eye, numerous small brownish patches of pigment were visible. They were marked by the brownish hue in contradistinction to the black color of pigment usually present in the pigment frame or in overpigmentation in the atrophic patches of old choroiditic lesions. The distribution was characterized by the fact that most of the patches were located in the macular region and there were only a few patches in the periphery. The patches were small, round or oval, without sharp outlines. In each eyeground one pigmented stripe was visible; in the course of the stripe, the retinal vessels were both underbridged and overbridged, denoting that the stripe was located in different layers of the retina.

COMMENT

It is important to note that in both cases there were a sudden onset, bilateral involvement, low grade uveitis and detachment of the retinas which healed without surgical intervention. The detachments in each case were rather extensive, and no holes or tears were present. The clinical course, however, differed widely in the two cases. In the first, the patient had "grip" before the onset of the ocular symptoms. The retraction of the iris-lens diaphragms and the long-lasting and extreme hypotonia warranted an unfavorable prognosis from the start. After reattachment of the retinas, the acute inflammatory symptoms continued and gained in severity. The deposits increased in number and size, and the seclusion membrane became thicker and vascularized, finally leading to the formation of complicated cataracts. The uveitis presented

itself in a most severe form, a type which is usually encountered in the group of sympathetic uveitis or inflammations closely related to it by their severity.

In the second case the inflammatory symptoms of the uvea were distinctly milder. In the early stages there were no deposits discernible, not even under slit lamp examination. The picture was within the framework of fibrinous iritis and was accompanied by deposits only later. After reattachment of the retinas, the entire inflammation subsided, the hypotonia disappeared and normal vision in one eye and almost normal vision in the other resulted. The retinal changes were similar in the two cases. In the second case the changes consisted of pigment proliferation within the retinas; in the first case, in addition to the proliferation, delicate, pointlike, white atrophic patches were noted in the choroid of each eye.

The sudden bilateral appearance in young persons of uveitis complicated with extensive retinal detachment markedly circumscribed the pathologic process in these cases. The careful clinical and laboratory analyses did not reveal any data of value concerning the cause of the disorder. In the first case the clinical appearance of the uveitis was of a much more severe type and of much longer duration than in the second.

Doubtlessly uveitis complicated with detachment with sudden onset and simultaneous appearance in each eye represents an entity in itself. Next to the bilateral appearance, the usual location in the lower part of the retina and the absence of holes or tears are significant. The latter condition is a usual finding in the inflammatory type of separation of the retina. But other types of inflammatory detachment may show tears occasionally, as I had occasion to observe in a case of unilateral tuberculous iridocyclitis in a young person, in which case, about three months after the acute iridocyclitis, a large detachment with a semi-circular tear presented itself, and diathermic coagulation of the tear produced a perfect and lasting result. In bilateral uveitis associated with detachment there seems to be no interruption in the continuity of the retina, and, accordingly, the healing and reattachment are achieved without surgical intervention. The usual unilateral inflammatory type of detachment, like that caused by scleritis or tuberculous choroiditis, is present and makes its appearance almost simultaneously in each eye.

The Japanese author Harada was the first, in 1926, to classify this group of conditions as a separate disease entity. Important are the bilateral appearance, the acute onset with headaches, occasional nausea and vomiting and the low grade inflammatory changes in the anterior segment of the uvea (or possibly, complete absence). Ophthalmoscopic examination reveals opacities of the vitreous and a diffuse

yellowish color of the retina with papillitis-like changes of the disk. After a few weeks extensive and bilateral detachment is noticeable and is followed by spontaneous healing and surprisingly good function, considering the pathologic involvement previously present. The end result presents depigmentation with pigment proliferation.

Takahashi¹⁰ used inoculation of the vitreous fluid in rabbits. The fluid did not contain pathologic micro-organisms. With intracisternal inoculation, in 2 of 5 cases he produced a descending optic neuritis and uveitis. The inoculation of the cerebrospinal fluid of the patient into the vitreous of rabbits produced inflammation in 1 of 4 cases. On inoculation of the cistern with brain tissue of infected animals, optic neuritis and iridocyclitis were produced; the direct inoculation into the eye produced inflammation of each eye by the route of the optic nerve. According to the results of inoculation, the disease should be classified as infectious, belonging in the category of sympathetic and herpetic lesions. Tagami's¹¹ inoculation of the vitreous of rabbits with subretinal fluid similarly resulted in uveitis, which histologically resembled sympathetic uveitis with the exception of giant cells. The infiltration in the anterior parts of the uvea was more marked; the infiltration of the choroid consisted of large round mononuclear elements. Furthermore, detachment of the choroid and retina through serofibrinous exudate and degeneration of the pigment epithelium with almost negligible changes within the retina took place. Accordingly, the disease, which seems to be more prevalent in Japan, should be classified with the virus group of diseases.

Contrary to these views are the results of Oka-Mura¹² who reported about 2 cases in his first, and 16 cases, in his second publication. The slit lamp revealed in the first case the presence of miliary nodules in the iris in large numbers, and a few nodules were discernible in the second case. Secondary glaucoma necessitated the performance of iridectomy in the first case, and histologic examination of the iris showed giant cells of Langhans type and therefore a similarity with sympathetic or tuberculous structure. In the second case the histologic findings should be analogous with those of beginning sympathetic lesions. Roentgenograms of the lungs, the sedimentation test and the general examination revealed tuberculosis. In the second publication Okamura considered all the cases of tuberculous origin. The nodules in the iris, the disseminated atrophy of the choroid, the perivascularitis of the retina and the bleeding into the vitreous are manifestations of that disease.

10. Takahashi, M.: *Acta Soc. ophth. jap.* **34**:33, 1930.

11. Tagami, K.: *Acta Soc. ophth. jap.* **35**:114, 1931.

12. Oka-Mura: *Acta Soc. ophth. jap.* **41**:679, 1937; **42**:196, 1938.

The discrepancy in the mechanism between virus disease and tuberculous origin is widespread and cannot be answered at present dogmatically. Further observation of similar cases will help greatly to solve the problem.

The clinical analysis shows that in smaller details there is a certain variation in the clinical picture presented. In the case of Salus the deposits and the nodules of the iris indicate a more active participation of the anterior segment of the uvea, a circumstance which could be interpreted in the light of a tuberculous origin. Salus was not inclined to consider it as such but emphasized the possible infectious character, citing the circumstance that the sister living with the patient suffered from a similar severe type of uveitis two months later. The case of Horay is more complex and therefore more difficult to classify. The absence of inflammatory symptoms in the anterior part, the bilateral (but not simultaneous) detachment with spontaneous healing and the resulting changes of the fundi are arguments in favor of classifying the condition as Harada's disease. The presence of marked scleritis and the development of a detachment, on the other hand, would place it in the group of cases of scleritis associated with retinal separation. In this group even repeated detachments are possible phenomena, as the recent publication of Zenker¹³ shows.

In the chapter on inflammatory detachments, the cases of bilateral simultaneous detachment accompanying uveitis doubtlessly represent a distinct and separate entity. The clinical picture differs from that of idiopathic detachment by the absence of holes and tears and is closely allied with that of unilateral detachment associated with scleral inflammation by the tendency to spontaneous healing. The depigmentation of the fundus and the proliferative pigment changes after healing has taken place are further characteristics of the entity. The primary cause leading to detachment is the uveitis, which seems to differ in intensity and expansion, as demonstrated by the severity in my first case and the relatively mild course of the second one. Similarly, in the cases published in the literature the uveitis is of various degrees; in some cases only the posterior segment of the uvea is involved, and in other cases the uveitis extends into the anterior segment. The inflammation of the anterior segment results in symptoms presenting various forms and degrees of inflammatory reaction. The underlying pathologic explanation is not held unanimously and varies all the way from that of a virus disease to a possible tuberculous origin.

31 Lincoln Park.

13. Zenker, C.: *Klin. Monatsbl. f. Augenh.* **102**:429, 1939.

RELATION OF DYNAMIC TO STATIC REFRACTION IN PRESBYOPIC PATIENTS FORTY THROUGH FIFTY YEARS OF AGE

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Although it is commonly recognized among ophthalmologists that the use of a cycloplegic drug is a necessity in the refraction of all persons who have not reached the age of presbyopia, yet it cannot be said that any truly general rule exists with respect to the use of cycloplegics in the refraction of presbyopic patients.

Many refractionists regard it as unnecessary to inconvenience their patients by the administration of a cycloplegic when the presence of presbyopia makes manifest refraction feasible. Others, of no less repute, do not believe that refraction can be done with scientific precision for even the most presbyopic person without cycloplegia.

The purpose of this study was to make a careful measurement of the refraction under dynamic and under static conditions and to compare the results in order definitely to demonstrate whether dynamic methods can be expected to uncover the full static correction for the average patient in the early stages of presbyopia.

The material for this research consisted of 100 persons with early presbyopia from 40 through 50 years of age who applied to the ophthalmic clinic of the Indianapolis City Hospital for refraction. When each patient was first seen, the refraction was determined by the manifest method by means of fogging. Then eucatropine hydrochloride in a 3 per cent solution was instilled into each eye every five minutes until a total of 5 drops (or more if required to obtain mydriasis) was administered. Thirty minutes after the instillation of the last drop refraction was done by retinoscopic and by trial case examination (retinoscopic examination alone being done for 25 of the 100 patients). At least seven days later each patient returned to the clinic after having instilled into his eyes at intervals of ten minutes a total of 10 drops of a 4 per cent solution of homatropine hydrobromide. Refraction was then done by retinoscopic and by trial case examination. No less

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then seven days after refraction with homatropine a postcycloplegic examination was made, at which time the addition required for near vision was determined. After refraction with homatropine, the far point of 91 of the patients was measured after a + 3 diopter sphere had been added to the infinity correction. The near and far points of 36 persons were both thus measured after manifest, eucatropine and homatropine refractions.

EXPERIMENTAL DATA

Refraction with homatropine is accepted as being the most accurate of the three methods. Therefore, the results of refraction by the manifest method or by retinoscopic and by trial case examination with the eye under the influence of eucatropine are directly compared with the results of refraction with the eye under the influence of homatropine.

The relation of refraction by the manifest method and with eucatropine to refraction with homatropine is of interest in the extent to which the algebraic sum of the sphere and cylinder in each case agree and in the degree of accord between the amount of cylinder and the position of the cylinder axis as found by the several methods. Of the two items, the latter is much the more important, for, after refraction with homatropine it is usually necessary to deduct a portion of the sphere in order to allow the accommodation a small amount of latitude in infinite vision. Therefore, the spherical element in the prescription is, in the age group of this study, of no great significance so long as it reasonably approximates the true sphere as uncovered by refraction with the use of a cycloplegic. Of much more significance is the algebraic sum of the sphere and cylinder, but this statistic, too, is influenced by possible deductions from the prescription. Hence, the chief source of information in the comparison of the three methods of refraction must be the statistics which concern the cylinder. Analyses of the algebraic sum of the sphere and cylinder and of the cylinder only will be presented, but all conclusions will be based entirely on the latter. The results with the manifest method are shown in the following outline:

Algebraic Sum of Sphere and Cylinder

Increase over that found with refraction with homatropine in 61 of 200 eyes,
or 30.5 per cent

Average increase, 0.381 diopter

Decrease below that found with refraction with homatropine in 110 of 200
eyes, or 55 per cent

Average decrease, 0.614 diopter

No change from that found with refraction with homatropine in 29 of 200
eyes, or 14.5 per cent

Amount of Cylinder and Deviation of Axis

Increase in the amount of cylinder over that found with refraction with homatropine in 36 of 200 eyes, or 18 per cent

Average increase, 0.317 diopter

Decrease in the amount of cylinder below that found with refraction with homatropine in 46 of 200 eyes, or 23 per cent

Average decrease, 0.692 diopter

Deviation of the cylinder axis in 87 of 200 eyes, or 43.5 per cent

Average deviation, 28.44 degrees

No change in the amount of cylinder or in the axis in 47 of 200 eyes, or 23.5 per cent

Cylinder uncovered by refraction with homatropine which was not detected by manifest refraction in 25 of 200 eyes, or 12.5 per cent

Average amount of latent cylinder, 0.415 diopter

Cylinder uncovered by manifest refraction which could not be detected by refraction with homatropine in 24 of 200 eyes, or 12 per cent

Average amount of false cylinder, 0.468 diopter

Deviation of the cylinder axis the only change in 22 of 200 eyes, or 11 per cent

The most striking fact which these data bring out is the high incidence of increase in the refractive error as determined by the manifest method. The algebraic sum showed such an increase in 30.5 per cent of all eyes examined, while the amount of cylinder was increased in 18 per cent. The average increase in the amount of cylinder, 0.317 diopter, is significant. This phenomenon may be accounted for by the fact that refraction by the manifest method is performed with the vision fogged, the correction accepted being that which permits the accurate discrimination of the letters in the 6/6 line with the greatest amount of plus lens. Under such circumstances it is conceivable that certain persons with good visual acuity, even with an increase in the amount of cylinder of 0.317 diopter, will readily be able to recognize the test letter even though the retinal image is not perfectly sharp.

A decrease in the amount of the cylinder occurred in 23 per cent of the eyes examined. As a decrease is rather the expected variation, it is surprising that its occurrence exceeded that of an increase by only 5 per cent of the eyes refracted. However, as might be expected, the average amount of decrease exceeded that of increase by better than 2 to 1. Certainly a cylinder which is estimated 0.692 diopter short of the true correction cannot be considered as representing excellence in refraction.

Forty-three and one-half per cent of all eyes examined showed the cylinder axes to have deviated an average of 28.44 degrees from their location at cycloplegic refraction. This constitutes a high percentage

of failure, for no refractionist cares to acknowledge an inability to locate the cylinder axis within such wide limits. Five degrees might be tolerated, but hardly 28!

Of the total number of eyes, 12.5 per cent showed a failure of the manifest method to uncover cylindric error of an average amount of 0.415 diopter, while 12 per cent showed the presence of cylindric error averaging 0.468 diopter which could not subsequently be confirmed by cycloplegic examination. Thus, the correction of the cylindric error was a complete failure in 24.5 per cent of all eyes refracted by the manifest method.

Not even in one fourth of the eyes refracted was the manifest method successful in revealing the true amount of cylinder and its axis. Such accuracy was found in only 23.5 per cent of the 200 eyes.

The results of refraction with eucatropine are shown in the following outline:

Algebraic Sum of Sphere and Cylinder

Increase over that found with refraction with homatropine in 84 of 200 eyes, or 42 per cent

Average increase, 0.331 diopter

Decrease below that found with refraction with homatropine in 82 of 200 eyes, or 41 per cent

Average decrease, 0.462 diopter

No change from that found with refraction with homatropine in 34 of 200 eyes, or 17 per cent

Amount of Cylinder and Deviation of Axis

Increase in the amount of cylinder over that found with refraction with homatropine in 25 of 200 eyes, or 12.5 per cent

Average increase, 0.338 diopter

Decrease in the amount of cylinder below that found with refraction with homatropine in 44 of 200 eyes, or 22 per cent

Average decrease, 0.477 diopter

Deviation of the cylinder axis in 64 of 200 eyes, or 32 per cent

Average deviation, 38.81 degrees

No change in the amount of cylinder or in the axis in 22 of 200 eyes, or 11 per cent

Cylinder uncovered by refraction with homatropine which was not detected by refraction with eucatropine in 33 of 200 eyes, or 16.5 per cent

Average amount of latent cylinder, 0.404 diopter

Cylinder uncovered by refraction with eucatropine which could not be confirmed by refraction with homatropine in 17 of 200 eyes, or 8.5 per cent.

Average amount of false cylinder, 0.382 diopter

Deviation of the cylinder axis the only change in 22 of 200 eyes, or 11 per cent

As is the case with the manifest method, there was a high incidence of increase in the refractive error when eucatropine was used. The algebraic sum showed an increase in refractive error in 42 per cent of the eyes examined. This is 11.5 per cent more than with the manifest method. On the other hand, the amount of the cylinder showed an increase in only 12.5 per cent of the eyes as compared to 18 per cent with the manifest method.

Decrease in the amount of the cylinder occurred in 22 per cent of the 200 eyes, which is 1 per cent less than the figure obtained by the manifest method. This, plus the fact that the average amount of decrease was distinctly less (0.215 diopter) than that found under refraction by the manifest method, suggests that the refraction with eucatropine is regularly uncovering a greater proportion of the error than the manifest method. Nevertheless, 0.477 diopter represents too great a deficiency in the amount of the cylinder to be consistent with true refractive accuracy.

Deviation of the cylinder axes occurred in 32 per cent of 200 eyes, representing a reduction of 11.5 per cent from that found with the manifest method. The average deviation is, however, 10.37 degrees greater with refraction with eucatropine. Although the latter method does represent some improvement on this point, the incidence of deviation is far too high and the average deviation of 38.81 degrees is intolerable to most patients.

Refraction with eucatropine failed to uncover cylindric error of an average amount of 0.404 diopter for 16.5 per cent of the 200 eyes examined, while for 8.5 per cent the presence of cylindric error averaging 0.382 diopter was uncovered which could not subsequently be confirmed by cycloplegic examination. This represents complete failure in correcting the cylindric error in a total of 25 per cent of all refractions with eucatropine. The average amount of the cylindric error not uncovered shows little variation from that reported with the manifest method, while the amount of false cylindric error uncovered shows a decrease in average amount of 0.086 diopter. As a whole, the record for refraction with eucatropine is no better than that for the manifest method of refraction in terms of complete failure in detecting cylindric error.

It would appear that, considering all of the data, the refraction with eucatropine represents some improvement over the manifest method in that it more closely approximates the refraction with homatropine cycloplegia. The explanation of this greater accuracy is to be found either in the fact that the use of eucatropine permits objective analysis by retinoscopic examination or in a cycloplegic property of the drug

or in a combination of both factors. No accurate information can be offered as to the true situation, but it is possible definitely to demonstrate that, contrary to the accepted pharmacology, eucatropine is a weak cycloplegic. Thirty-six patients, representing 72 eyes, had their puncta proxima¹ measured with a plus 3 diopter lens added to the distance correction after each refraction was performed. Comparison of the puncta proxima after refraction by the manifest method with that found after refraction with eucatropine gives evidence of the development of cycloplegia after the use of eucatropine in 46 of the 72 eyes, or in 64 per cent.

1. Increase in the total plus lens with recession of the puncta proxima 34 eyes, or 47.2 per cent
2. Increase in the total plus lens with no change in the position of the puncta proxima..... 10 eyes, or 14 per cent
3. No change in the total plus lens with recession of the puncta proxima 2 eyes, or 2.8 per cent

These data can leave no doubt that some degree of cycloplegia is induced by the use of eucatropine and plays a definite part in enhancing the efficacy of this type of refraction. The experiment, however, has not been controlled in such a fashion as to permit any deduction concerning the relative importance of cycloplegia as compared to objective examination with the retinoscope. It must, therefore, suffice that both factors are definitely present, their exact relation remaining, for the present, unknown.

Of considerable interest is the distribution of depth of presbyopia among the cases analyzed. The additions prescribed at the time of the postcycloplegic examination afford an accurate indication of the state of presbyopia in each case:

1. No addition required..... 13 per cent of cases
2. An addition no greater than 1 diopter required..... 26 per cent of cases
3. An addition of more than 1 diopter but no greater than 2 diopters required 45 per cent of cases
4. An addition greater than 2 diopters required..... 16 per cent of cases

It now becomes evident that the poor showing made by refraction by the manifest method and with eucatropine as compared to the results obtained with refraction with homatropine cannot be explained by the claim that the group of patients examined, having early presbyopia,

1. Mydriasis has little effect on the accommodation. As stated in a recent paper published in the ARCHIVES (An Evaluation of Homatropine-Benzedrine Cycloplegia, Arch. Ophth. 20:585-596 [Oct.] 1938), I could find no change in the position of the punctum proximum in 13 of 15 cases after mydriasis with benzedrine sulfate.

had too great an accommodative power, for 61 per cent of the 100 patients required an addition greater than 1 diopter. The patients requiring an addition of more than 2 diopters exceeded those who required no addition by 3 per cent, and only 26 per cent required an addition of less than 1 diopter.

Still another approach to the study of the relation of refraction by the manifest method and with eucatropine to refraction with homatropine involves an analysis of the distribution among the several classes of persons with presbyopia (as presented in the preceding outline) of refractions obtained by the first two methods which are identical with those obtained with the use of homatropine:

	Manifest Refraction		Eucatropine Refraction	
	No.	%	No.	%
Class 1.....	2	7.7
Class 2.....	2	3.8
Class 3.....	5	5.5	7	7.7
Class 4.....	3	9.4	5	15.6

The occurrence of identical refractions in classes 1 and 2 is so rare as to make the percentage figure of little value. Class 3 gives a percentage accuracy for refraction by the manifest method of 5.5 and for refraction with eucatropine of 7.7. Class 4 shows a percentage accuracy for the manifest method of 9.4 and for refraction with eucatropine of 15.6.

Thus, there is established a definite trend toward greater accuracy with both manifest and eucatropine refraction the more advanced the presbyopia. It also appears that eucatropine refraction is somewhat more accurate than the manifest refraction and that this differential in accuracy increases with increasing depth of presbyopia. However, in class 4 (those persons requiring additions in excess of 2 diopters) absolute exactness in refraction as determined by comparison with homatropine refraction was attained in the case of the manifest examinations in only 9.4 per cent of the eyes refracted and in the case of eucatropine refraction in 15.6 per cent. This is true despite the common conception that the accommodation will not greatly interfere with the determination of the refraction by manifest methods after the age of 45. The percentages noted are high for the group as a whole, for class 4 constitutes only 16 per cent of the 100 patients. For the entire series of 200 eyes, the refraction with the manifest method showed a percentage of efficiency of 5 and with eucatropine a percentage of 7—rather too low to encourage the refractionist to dispense with cycloplegia in dealing with patients in this age group.

SUMMARY

A study is here reported of 100 patients from 40 through 50 years of age, refraction for each of whom was done by the same person by the fogging method of manifest refraction, by retinoscopic and by trial case examination after the instillation of eucatropine hydrochloride and by retinoscopic and by trial case examination after the instillation of homatropine hydrobromide, the latter being followed by postcycloplegic examination.

Eighteen per cent of the eyes examined by the manifest method and 12.5 per cent of those examined under the influence of eucatropine showed an increase in the amount of the cylinder over that detected by refraction with homatropine.

Decrease in the amount of the cylinder occurred in 23 per cent of eyes examined by the manifest method and in 22 per cent of those examined under the influence of eucatropine, the average amount of decrease being distinctly less with eucatropine.

Deviation of the cylinder axis occurred in 43.5 per cent of eyes examined by the manifest method and in 32 per cent of those examined under the influence of eucatropine, the average amount of deviation being greater with eucatropine.

Complete failure to detect cylindric error (evidenced either by failure to detect cylindric error which existed under refraction with homatropine or by detection of cylindric error the existence of which could not be confirmed by such refraction) occurred in 24.5 per cent of eyes examined by the manifest method and in 25 per cent of those examined under the influence of eucatropine. The amount of latent or false cylindric error was slightly less with eucatropine refraction than with the manifest method.

Comparison of the punctum proximum after manifest refraction with that after refraction with eucatropine demonstrated the development of cycloplegia in 64 per cent of the 72 eyes for which data were available.

In cases of advanced presbyopia, in which the addition required was in excess of 2 diopters, there was failure exactly to duplicate the homatropine refraction in 90.6 per cent of the eyes examined by the manifest method and in 84.4 per cent of the eyes refracted under the influence of eucatropine.

CONCLUSIONS

1. Refraction by the manifest method and with eucatropine does not compare favorably with refraction with homatropine because:

(a) There are sizable variations in the amount of cylindric error that each method uncovers.

(b) There is too frequent occurrence of variation in the location of the cylinder axis over a wide arc.

(c) There is complete failure to detect the cylindric error in a large proportion of the eyes examined.

2. Because of slightly less pronounced variations as enumerated, refraction with eucatropine appears to be slightly more efficient than with the manifest method.

3. Eucatropine is demonstrated to be a weakly cycloplegic as well as a mydriatic drug.

4. Refraction by the manifest method and with eucatropine, even in cases of advanced presbyopia, does not reveal the full cycloplegic refraction except for a small percentage of the eyes examined, the percentage being greater in the case of refraction with eucatropine.

5. All evidence indicates that for more optically precise refraction presbyopic patients in the fifth decade of life should be examined under homatropine cycloplegia rather than by the manifest method or after the use of eucatropine.

A NEW CORNEOSCLERAL SUTURE

JOHN M. McLEAN, M.D.

BALTIMORE

In 1894 Kalt¹ described the first corneoscleral suture. This consisted of two vertical bites, one in the cornea and one in the sclera, with 1 mm. between, in which the section was made. He later modified this procedure, inserting the scleral bite horizontally to make a T-shaped suture. Liégard² further modified Kalt's suture by placing both bites horizontally. This modification is often used today.³ There are certain drawbacks to this type of corneoscleral suture. The wound is not covered by conjunctiva, although such protection may be partially obtained by a sliding Van Lint flap.⁴ Instead of providing perfect approximation, the vertical arms of the suture allow a certain amount of lateral displacement, and because they run up over the lips of the wound they tend to produce inversion if tied too tightly. The latter fact may predispose to epithelial downgrowth as well as to imperfect union. Suarez de Mendoza⁵ in 1892 tried to avoid some of these defects by making a preliminary incision from without in clear cornea and inserting the ends of a double-armed suture in either lip. Section was then completed through this prepared suture. While obviating some of the difficulties of Kalt's suture, this method had all the inherent drawbacks of a completely intracorneal section with unprotected edges and never became popular. Baldino⁶ modified this method by inserting the needle, making the section up to it and completing the section by

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

1. Kalt, E.: On the Corneal Suture in Cataract Extraction, *Arch. Ophth.* **23**:421, 1894.

2. Liégard, H.: Une modification au procédé de suture de la cornée dans l'opération de la cataracte, *Ann. d'ocul.* **149**:119, 1913.

3. (a) Ellett, E. C.: Use of the Suture in Extraction of Cataract, *Arch. Ophth.* **17**:523 (March) 1937. (b) Stallard, H. B.: A Corneo-Scleral Suture in Cataract Extraction: Its Technique and Advantages, *Brit. J. Ophth.* **22**:269, 1938.

4. Leech, V. M., and Sugar, H. S.: Reduction of Post-Operative Complications in Cataract Operations, *Arch. Ophth.* **21**:966 (June) 1939.

5. Suarez de Mendoza, F.: Nouveaux faits de suture de la cornée dans l'extraction de la cataracte, *Bull. et mém. Soc. franç. d'opht.* **10**:63, 1892.

6. Baldino, S.: Sutura corneale perfezionata, *Rassegna ital. d'ottal.* **4**:538, 1935.

dissecting out the needle and then pulling the suture through. Müller⁷ in 1903 dissected a corneal pocket from below upward, placed two sutures in the edges of the pocket and then made a section up to the base of this corneal flap. Walker⁸ in 1929 reversed this procedure, dissecting the corneal pocket from above downward. Verhoeff⁹ in 1927 described two types of corneoscleral sutures with protection of the top of the wound by a sliding flap. The first suture was devised in an attempt to improve on Kalt's original suture by "sawing" a tract with the suture itself before making the section. The other suture was theoretically more perfect; a single vertical bite is taken at the limbus above, the section is made through it and the tract is then rethreaded after the eye is open. The last step presents obvious difficulties with unruly patients or when vitreous presents on section. Finally, in 1938 Lindner,¹⁰ finding Verhoeff's sutures too difficult, reintroduced Suarez de Mendoza's method, placing the suture back in the sclera and using the hair of Japanese women instead of silk.

The ideal suture for closing cataract wounds should have certain definite characteristics. It should be inserted in solid corneal and scleral tissue and not in loose, yielding conjunctiva. It should be placed before the section is made and should not require extensive manipulations after the eye is opened. It should go through, not over, the lips of the wound, so as to give firm closure without danger of inversion of the edges, and it should be so placed that when tied it will bring the tissues back to exactly the preoperative position. The entire wound should be covered by conjunctiva as an added protection and better surgical closure. The method should not be too complicated for the average surgeon.

A suture which seems to meet these criteria has been developed at the Wilmer Ophthalmological Institute in the past year. The technic follows:

A small conjunctival flap is dissected down to the limbus around the entire upper half of the eye. At the base of this flap a small slot is made with a Lunds-gaard knife (figs. 1 *A* and 2 *A*) about half-way through toward the anterior chamber. A cataract knife, keratome or small sharp scalpel could be used for the same purpose. A fine black silk suture on Kalt's corneal needle is then run through the base of the conjunctiva, reversed, and run through both the scleral

7. Müller, L.: Ein Operationsverfahren für komplizierte Stare und luxierte Linsen, *Klin. Monatsbl. f. Augenh.* **41**:11, 1903.

8. Walker, C. B.: Exactly Appositional Sutures in the Cataract Operation, *Tr. Am. Ophth. Soc.* **27**:51, 1929.

9. Verhoeff, F. H.: A Corneo-Sclero-Conjunctival Suture in Operations for Cataract, *Tr. Am. Ophth. Soc.* **25**:48, 1927.

10. Lindner, K.: Ueber Abänderungen der intracapsulären Staroperation, *Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch.* **52**:392, 1938.

and the corneal lips emerging in clear cornea just below the base of the conjunctival flap (figs. 1 *B* and 2 *B*). The suture is thus placed so that when it is later tied it will bring the lips of the wound back to their original position before the eye is opened. The suture is then pulled out of the slot with a blunt iris hook (figs. 1 *C* and 2 *C*) and the loops laid aside to give room for the cataract knife. Section is made under the flap with a knife (fig. 1 *D*), which emerges at the base of the slot between the arms of the suture. Such a section is not as difficult as it sounds and has been successfully performed by inexperienced house officers on first trial. Extraction is performed in the usual way and the suture

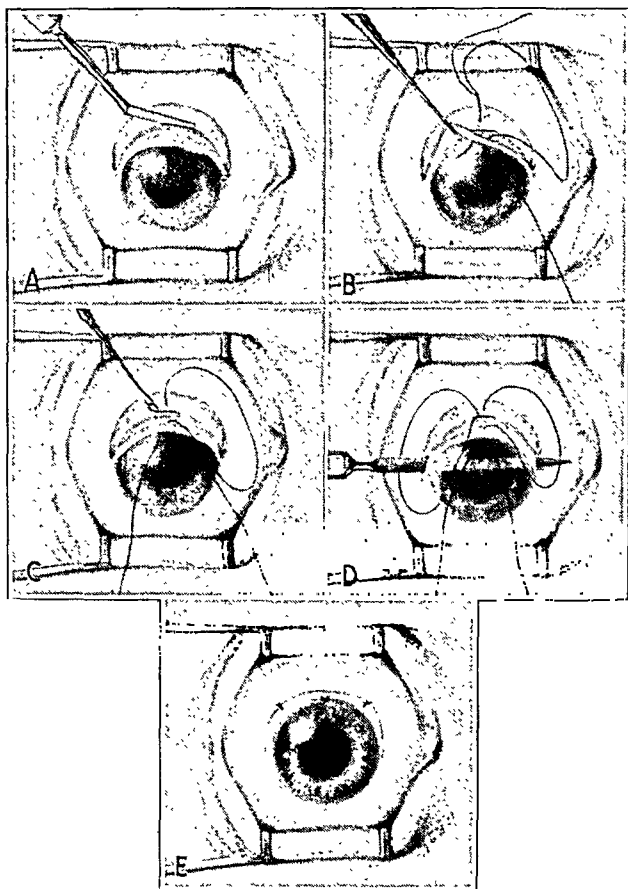


Fig. 1.—Technic of operation with the corneoscleral suture. In *A*, a small groove is made beneath the conjunctival flap with the Lundsgaard knife. In *B*, the suture is placed through the scleral and the corneal lips of the groove. In *C*, the suture is drawn out of the groove with a blunt iris hook. In *D*, section is made with a cataract knife, which emerges through the groove above. In *E*, the corneoscleral suture is tied and two additional conjunctival flap sutures are placed at the end of the operation.

pulled taut and tied at the end of operation (figs. 1 *E* and 2 *D*). If irrigation or extensive toilet of the wound is necessary, the suture may be tied before, making these maneuvers much safer. Further sutures may be placed in the edges of the conjunctival flap if this seems advisable to hold it in place, but firm closure of the wound is maintained by the corneoscleral suture. Although one such suture

gives excellent closure with perfect apposition, two, or even three, may be used when the operator is particularly apprehensive about a patient's postoperative behavior. The suture is not at all irritating and can be left in place for some time. Although Ellett^{3a} advised removal of corneoscleral sutures on the third or fourth day, and Stallard^{3b} on the fourteenth day, it has been the general practice in the institute to remove these sutures between the tenth and the twelfth day.

RESULTS

Ellett,^{3a} Stallard,^{3b} Lindner¹⁰ and many others have pointed out the advantages of corneoscleral sutures: (1) firm closure with prompt healing, (2) reduction of postoperative hyphema, (3) less risk of

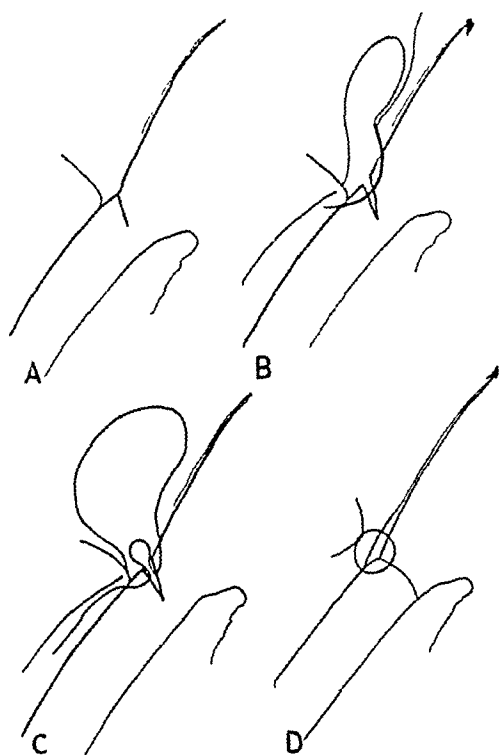


Fig. 2.—A, schematic cross section of the prepared slot behind the conjunctival flap. B, schematic cross section of the insertion of the corneoscleral suture. D, schematic cross section of the corneoscleral suture at the end of the operation. C, schematic cross section of the suture being drawn out of the groove.

prolapse of the iris or vitreous, (4) safer repair of prolapse of the iris if it should occur, (5) less postoperative astigmatism, (6) better closure if vitreous presents at operation, (7) earlier reformation of the anterior chamber and (8) greater freedom of movement for elderly patients and less danger for uncooperative ones.

Only Leech and Sugar have presented statistics to bear out these contentions. In their series of 450 cases, the incision was closed without a suture in 150, with conjunctival flap sutures in 150 and with a corneoscleral suture of a modified Liégard type in 150. Prolapse of the iris, vitreous or both and delayed reformation of the anterior chamber

occurred most frequently in the cases in which no suture was used and least frequently in those in which the corneoscleral suture was employed. The incidence of postoperative hyphema was least with the corneoscleral suture, but, interestingly enough, greatest with the conjunctival flap suture. This is probably a coincidence, but it might be a result of firm closure of the conjunctival wound with subsequent gaping of the unsupported corneoscleral incision beneath it under pressure of newly formed aqueous. The average amount of postoperative astigmatism was greatest when no suture was used and least when the corneoscleral suture was employed. These operations were all performed in the same institution but by various surgeons.

The series of cataract extractions here reported are those done by me during my year as resident surgeon at the Wilmer Ophthalmological Institute. They provide a smaller number of cases but a series in which

Comparison of Results with Both Types of Sutures

	Corneoscleral Suture	Conjunctival Suture
Total number of cases.....	110 (100%)	64 (100%)
Delayed reformation of anterior chamber or delayed closure of wound.....	4 (3.6%)	9 (14.1%)
Hemorrhage into anterior chamber.....	4 (3.6%)	12 (18.7%)
Choroidal hemorrhage	0	2 (3.1%)
Prolapse or incarceration of iris.....	3 (2.7%)	4 (6.3%)
Average cylinder	1.32	2.56

all operations were performed by the same surgeon under similar circumstances and with similar technic except for the use of either the conjunctival or the corneoscleral suture. Similar results have been obtained with this corneoscleral suture by other surgeons at the institute, but in order to keep all other factors constant only my results are analyzed.

In all, 174 extractions were performed. One hundred and twenty-nine were for ordinary senile cataracts and 45 for complicated cataracts of various types. Twenty-one were extracapsular extractions and 153 intracapsular extractions. In 99 cases one corneoscleral suture, as previously described, was used; in 11, two such sutures were used. In the remaining 64 cases closure was done with multiple conjunctival flap sutures. There are therefore available for comparison 110 cases in which the corneoscleral suture was used and 64 cases in which only the conjunctival suture was used. This comparison, with complications and the final amount of astigmatism, is summarized in the accompanying table.

Delayed union of the lips of the wound or delayed reformation of the anterior chamber was markedly reduced by the use of the corneo-

scleral suture (3.6 per cent contrasted with 14.1 per cent). The most striking improvement attributable to the use of this type of suture was the reduction of hemorrhage into the anterior chamber from 18.7 to 3.6 per cent. This complication was generally encountered around the fourth or fifth day; it was sometimes related to undue trauma on the part of the patient and more often appeared spontaneously, apparently without definite reason. All instances of seepage of blood into the anterior chamber were recorded, no matter how slight. Although the two deep choroidal hemorrhages occurred after the use of conjunctival flap sutures, this cannot be considered significant. Small incarcerations of the iris occurred in 3 cases (2.7 per cent) in which the corneoscleral suture was used, in 2 of them after extraction with round pupils, but none was severe enough to require repair. Prolapse or incarceration of the iris was found in 4 cases (6.3 per cent) in which conjunctival suture was used, in 3 of them after extractions with full iridectomy. One of these was only a minor incarceration, but the other three were true prolapses and were repaired. It is interesting to note that the average cylinder when the corneoscleral suture was used was almost exactly half that found when the conjunctival suture (1.32 diopters contrasted with 2.56 diopters) was employed.

SUMMARY

A new type of corneoscleral suture is described. Use of this suture has resulted in a marked reduction of gaping wounds, postoperative hyphema, prolapse of the iris and postoperative astigmatism.

THE ORTHOPTIC TECHNICIAN AS AN AID TO THE OPHTHALMOLOGIST

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SAN FRANCISCO

The function of the orthoptic technician is to aid the ophthalmologist in establishing habits of correct binocular vision. In establishing any habit, the desired activity must first be physically possible for the patient; it must be within his capacity to perform the act. It is not always possible to determine with a single lesson whether the function of binocular vision is impossible or merely in abeyance. Once the possibility of binocular vision is established, however, the steps which follow are a typical educational process and constitute the second aspect of habit training.

The ophthalmologist may conveniently think of his patients for orthoptic training as falling into two classes: those on whom he expects to operate and those for whom he does not expect to use operative procedure.

For the first group he may describe preoperative and postoperative training. Preoperative orthoptics may begin with the treatment of amblyopia ex anopsia. Orthoptics may be made useful both by office treatments consisting of flashing and movement on orthoptic machines, monocular reading with graded print on the metronoscope and similar devices to stimulate the amblyopic eye, and by suggestions to the patient and family for home activities that will help maintain interest and cooperation through the often tedious period of reducing the amblyopia.

Regular training can begin as soon as the visual acuity is sufficient for the patient to see the targets, if necessary with especially favorable lighting, which may be brighter for the amblyopic eye.

The first thing which the orthoptist measures is anomalous correspondence. It is not within my province to define or explain this still moot condition, but to the technician it manifests itself on the synoptophore as a use of the eyes, which, if not corrected, makes subsequent training fail in its effect. It usually takes from ten to twenty lessons to establish normal correspondence, and once it is corrected it shows little tendency to relapse. Very often correcting the correspondence will result in a slightly improved position of the eyes.

If anomalous correspondence is not present, or after it has been corrected, the next step is the development of fusion with amplitude, if

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possible to include the position of balance while simple targets are being fused. Some patients lack fusion ability; they have no fusion sense. With the development of improved orthoptic instruments and technic, such persons seem to be fewer and fewer. Experienced technicians report that they have never seen a patient with true alternation; they all break down under training.

The first step in fusion training is to find out just the conditions necessary for fusion: the angle of fixation, the lighting and the type of target to which the patient responds the best. Patients often have idiosyncrasies regarding a target and will work better with one than with another of similar type. They will sometimes ask for hyperadjustments or even cycloadjustments, which are discarded as soon as training is begun.

A common obstacle to fusion is the crossing over, or suppression, of targets in whole or in part at just the point where fusion should take place. Such suppression acts like a scotoma and can be so located and mapped. Training will show a systematic reduction in the size of the scotoma. When the surrounding areas are successfully fused, the scotoma gradually disappears.

A second difficulty is extreme instability of fusion. The images fuse first in one position and then in another. The orthoptist watches the corneal reflexes and seizes favorable positions for stimulation until fusion becomes more marked within limited areas and there is a definite position from which to work.

Once fusion is established, the development of amplitude follows. It is usually somewhere at this point of the training that the ophthalmologist operates, if he plans to do so at all. The time element is often of importance, and the development of adequate amplitude may take several months of orthoptic training alone or may be beyond the power of this technic in specific cases.

Postoperative training should begin as soon as the eye is sufficiently healed to make it safe, usually within a week or ten days. The muscles and coordination are then still pliable, and much can be accomplished in establishing a correct position and in overcoming former faulty habits of use. The development of amplitude is continued, with training in depth perception. Home exercises are taught, together with bar reading and other checks that will help the patient to understand how well his eyes are functioning.

Among nonoperative conditions, there are two special types that I wish to discuss: accommodative squints and convergence insufficiencies.

Accommodative squints may or may not belong in the nonoperative category. If a person has no more than about 3 diopters of hypermetropia and can begin training before 7 years of age, the prognosis for cure by orthoptics is favorable. There are different definitions of

cure of accommodative squints, but to the orthoptist it means binocular vision without deviation, muscle balance for near and far vision with the cover test, and four lights for distant vision and four or five for near vision with the Worth four dot test. To achieve this objective, it is necessary to teach the patient to dissociate his accommodation and convergence. When he learns that by seeing blurred images he can keep his eyes straight and still fuse, he has accomplished an important step. A person with mild hypermetropia will often then accommodate and obtain clear vision without converging again. It is this type of patient who can be encouraged to go without his glasses, at first for an hour or so at mealtimes, when he can be corrected readily if his eye deviates without his realizing it. Of course if he has much astigmatism or too high a degree of hypermetropia, he will not get clear vision in any case. This is one reason why such persons must be selected with care in order to obtain good results by orthoptics alone.

The sequence of treatment for such persons begins exactly as for those who are to be operated on: correction of amblyopia; correction of anomalous correspondence, which is especially common in accommodative squints, and development of fusion. As fusion training proceeds, it also teaches the patient to appreciate diplopia, first with targets and later with reference to casual seeing. The use of small lights, colored glass and similar devices stimulates appreciation of diplopia. Home exercises with lights and the finger help speed up this phase of training. It is important to lay a thorough groundwork at this stage, for it is by means of diplopia that the patient learns when his eye is deviated. The development of amplitude to balance gives him the power to fuse; the development of diplopia gives him the key to maintaining fusion.

Experience has proved that the diplopia that appears as a stage of orthoptic training is not nearly so distressing as other types of diplopia. It is fleeting, tends to be vague and soon disappears in favor of fusion.

It takes from four to eight months to teach an intelligent child this correction of accommodative squint, and the results are usually gratifying. Treatment may be discontinued at any point that the results seem adequate to meet the situation, or it can be carried on until complete control is developed. In no case can worth while results be expected in less than three months with two or three treatments a week, and if it is unlikely that the patient will cooperate for this length of time it is not hopeful that success can be achieved by orthoptics alone.

Convergence insufficiencies yield well to orthoptic training. The typical subjects are young adults who train and cooperate well. They are usually willing to do home exercises as a supplement to office training. They develop amplitude rapidly, and when their margin of reserve

is adequate they find convergence easy and comfortable. Two to six weeks brings striking improvement in such cases.

Since orthoptics cannot correct anatomic anomalies, either congenital or acquired, it is of the greatest importance that the causation of the condition for which training is given shall be clear so that patients suitable for training are selected. It is often impossible to give a satisfactory prognosis until after two weeks of treatment, or even longer in difficult borderline cases.

Orthoptics is essentially a process of reeducation, subject to the limitations of educational procedure.

490 Post Street.

LINDAU'S DISEASE

REPORT OF SIX CASES, WITH SURGICAL VERIFICATION IN FOUR
LIVING PATIENTS

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In 3 cases a diagnosis of Lindau's disease¹ has been verified recently by craniotomy at the Toronto General Hospital, and in a fourth case, that of a blind sister of 1 of the patients in the aforementioned 3 cases, a cerebellar hemangioendothelioma was removed in Vancouver, B. C., in 1938. All 4 patients have since returned to their work. The mother in one of the two families involved died after an operation for tumor of the brain in 1918; the father in the other family died in 1916 of intracranial complications; both of them had been blind in one eye for five years before death. In all, 6 of the 11 members in two generations have been affected, and the surgical diagnosis has been confirmed by pathologic examination in 2 of the cases in which the patients are living.

Before 1928 fatal results were uniformly reported in the treatment of Lindau's disease. The earliest records have shown clearly the familial incidence of multiple angiomas involving the central nervous system, principally the cerebellum. The cerebrum, the spinal cord and the retina are of secondary importance to the cerebellum as sites for the angiomas that accompany Lindau's disease, according to statistics on fatal cases. Of less importance are cystic changes in kidneys, pancreas, epididymis, liver and bones and the finding of another variable factor, hypernephroma. It is to be noted that the fibrovascular layer of the retina was the site of Hippel's disease, or angiomatosis retinae. This layer is embryologically allied with brain tissue.

The oldest patient is reported as being 48 years of age,^{1a} but the condition is usually seen in the second or the third decade of life. It is slowly progressive, and in recent years in a few verified cases it has, it is hoped, been permanently arrested by extirpation, or when gross removal was impossible on account of the danger of damage to surrounding structures, by decompression, local diathermy and tapping, with the addition of irradiation.

Read at the Seventy-Fifth Annual Meeting of the American Ophthalmological Society at Hot Springs, Va., June 6, 1939.

1. Lindau, A.: *Acta ophth.* 4:193, 1927.

1a. The data on this patient are included in the condensed table of cases of Lindau's disease with surgical verification in living patients.

Dr. K. G. McKenzie, of the neurosurgical department of the Toronto General Hospital, surgically verified the diagnosis of Lindau's disease in 3 of these cases. The blind sister of 1 of the patients was able to resume her work at the Institute for the Blind after an operation in Vancouver, B. C., Canada, when a cerebellar hemangioendothelioma was removed by Dr. Turnbull² in 1938. In a series of 53 verified cerebellar tumors at the Toronto General Hospital there were 10 other cerebellar hemangiomas (1932-1938 inclusive); these did not have a hereditary relation with Lindau's or Hippel's disease.

The year 1928 may also mark the division of Lindau's disease into two categories, fatal and nonfatal, as it is evident that the widespread complications reported by the early writers will no longer be seen. The ophthalmologist is principally concerned with the second category, in which there is hope for recovery. The cerebellar tumors of Lindau's disease are found in about 1 of every 5 patients who suffer from angiomas of the retinae, or Hippel's disease. Angiomas of the retinae also has a familial incidence according to Usher,³ who in 1936 collected 119 cases, in 18.48 per cent of which the condition was inherited, 22 patients in 8 pedigrees being affected.

A review of the records of the complete syndrome of angiomas of the retinae plus cerebellar tumor shows less than 50 cases, and it may be doubted that the condition in those reported by Isnel,⁴ Davidoff,⁵ Wessely⁶ and others should be considered Lindau's disease, as a factor of heredity, angiomas of the retinae or cerebellar tumor is wanting; in other words, hemangioendothelioma alone is not Lindau's disease.

The cases of Lindau's disease in the second family reported on here may be considered as belonging to the list of cases of Lindau's disease in which diagnosis is yet to be confirmed. There is no record in case 4 of a retinal angioma, and the cerebellar angioma was lost at operation, when the small mural tumor disappeared in the sucker as the surrounding cyst was evacuated. The sister of the patient was completely blind before a hemangioendothelioma was removed from her cerebellum, and the father died of an intracranial complication after five years of blindness.

The failure to diagnose the condition in case 1 as Lindau's disease until after the sister's operation leads me to believe that blindness without a history of trauma, when associated with the factor of heredity,

2. Turnbull, F.: Personal communication to Dr. K. G. McKenzie.

3. Usher, C. H.: *Tr. Ophth. Soc. U. Kingdom* **50**:183, 1935.

4. Isnel, R.: *Bull. Soc. d'opht. de Paris*, December 1934, p. 652.

5. Davidoff, L. M.: *Am. J. Path.* **5**:141, 1929.

6. Wessely: *Klin. Monatsbl. f. Augenh.* **90**:95, 1933.

should be considered, for the purpose of diagnosis, as a possible equivalent of angiomatosis retinae. The essential cause of blindness in case 1 was obscure until the sister's operation, and even then confirmation was difficult by pathologic examination on account of the small size of the retinal tumor.

The successful treatment of angiomatosis retinae by radon seeds has been recorded by Foster Moore,⁷ although a large defect of the visual field resulted. It is hoped that secondary glaucoma and retinal

*Data on Ten Cases of Lindau's Disease with Surgical Verification
in Living Patients*

Reporter	Patient	Sex	Age	Hered- ity	Angio- matosis Retinae	Comment and Findings
Cushing and Bailey: Arch. Ophth. 57: 447, 1928; Tumors Arising from the Blood Vessels of the Brain, Springfield, Ill., Charles C. Thomas, Publisher, 1928	F. McA.	M	30	+	Yes	Cerebellar hemangioma
Møller: Acta ophth. 7: 244, 1930	O. C. H. J.	M	44	+	Yes; enucleation	Hyperplastic cerebellar capillary hemangioma
Viets: J. Nerv. & Ment. Dis. 77: 457, 1933	F. L. W.	M	48	No	Yes	Inoperable cerebellar tumor; cure with irradiation
Atkinson: Arch. Ophth. 7: 510 (April) 1932	J. R.	M	32	No	Yes	Angioma and cyst removed
Hope-Robertson: Australian & New Zealand J. Surg. 4: 55, 1934	O. F. D.	F	16	+(?)	Detachment	Hemangioendothelioma: treated by diathermy
MacDonald	Miss R.	F	23	+	Yes; blind	Cerebellar tumor; irradiation; eye enucleated
MacDonald	Mrs. S.	F	29	+	Yes	Hemangioendothelioma of cerebellum
MacDonald	A. P.	M	30	+	No	Cerebellar tumor and cyst; specimen lost
MacDonald	Mrs. T.	F	29	+	Blind	Patient operated on in Vancouver; cerebellar angioma
Casten: Personal communication to author; Am. J. Ophth. 16: 999, 1933	M	48	No	Yes	Retinal cyst and pontile angioma

detachment will be prevented by radiation of a degree intermediate to the reported extremes. Therefore, in treating the patient in case 2 four radon seeds were used, each of 0.7 millicurie, attached to a silk suture at intervals of about 4 mm., for seven days on the sclera opposite the tumor.

The terms Hippel's disease and Lindau's disease must be retained in describing conditions associated with the names of Hippel and Lindau, for while both conditions have a familial tendency, Hippel's disease may occur without complications. The neurosurgeon and the

7. Moore, R. F.: Tr. Ophth. Soc. U. Kingdom 53:215, 1933.

pathologist are more interested in syringomyelia, syringobulbia and cerebral cysts, which, in the absence of angiomatosis retinae, form part of the syndrome of Lindau's disease and have been reviewed by Wolf and Wilens,⁸ Brandt,⁹ Dandy,¹⁰ Sargent and Greenfield.¹¹ The cystic changes in the pancreas and the kidney are of interest chiefly to the pathologist.

REPORT OF CASES

Family R.—CASE 1.—Miss E. R., a white woman aged 23, a nurse, was referred to Dr. K. G. McKenzie on Feb. 3, 1936, by Dr. William S. Butler, of North Bay, Canada. She had suffered from headache in the region of the vertex and vomiting for one year, which was increasing in severity. Two years previously, on bending over, she had suddenly lost the sight of her right eye, and when she was seen shortly after this by Dr. C. Hill it was stated that the loss of vision was due to an intraocular hemorrhage. Her appendix had been removed on account of the vomiting. A tentative diagnosis of tumor of the brain was made, and she was admitted to the Toronto General Hospital on February 4.

Inquiry as to past illnesses and functional factors revealed nothing of importance. Physical examination gave essentially negative results, showing only a healed scar at McBurney's point and blood pressure of 90 systolic and 60 diastolic. Neurologic examination showed good intelligence, cooperation and memory. Vision in the right eye was 6/18; with glasses it improved to 6/6. There was no perception of light in the left eye. The visual field of the right eye was normal; the physiologic cup was filled. There was slight blurring of the borders of the disk. The right pupil reacted to light and in accommodation. The left pupil was irregular and showed ectropion of the uveal pigment with a dense opacity in the lens. The fifth, eighth, ninth, tenth, eleventh and twelfth nerves were normal. The patient was right handed, and the strength in each hand was good. Sensation to touch, light, heat and cold was normal. The reflexes were normal, except that the triceps reflex was not obtained. Stance was good, although the patient felt weak. Romberg's sign was negative. Slight staggering on walking was possibly due to weakness.

A provisional diagnosis was made of congenital aneurysm about the circle of Willis.

On Feb. 10, 1936, Dr. McKenzie did a first stage cerebellar exploration. The patient reacted poorly, and the pulse rate rose to 150. The arch of the atlas was removed.

On February 11 there was no vomiting, and the right disk was still swollen.

On February 24 Dr. McKenzie performed a second stage cerebellar operation. The patient again reacted poorly, but a vascular tumor, about the size of a large walnut, was found when the dura was opened. It occupied the lower half of the right cerebellar hemisphere and caused direct pressure on the medulla. A large venous channel, $\frac{3}{8}$ inch (0.3 cm.) in diameter, passed from the tumor to the midline to empty into the transverse sinus. It seemed impossible to remove the vascular tumor, so only decompression was done.

8. Wolf, A., and Wilens, S. L.: *Am. J. Path.* **10**:545, 1934.

9. Brandt, R.: *Arch. f. Ophth.* **106**:127, 1921.

10. Dandy, W. E.: *Arteriovenous Aneurysm of Brain*, *Arch. Surg.* **17**:190 (Aug.) 1928.

11. Sargent, P., and Greenfield, J. G.: *Brit. J. Surg.* **17**:84, 1929. Paton, L., and Williamson-Noble, F. A.: *Tr. Internat. Ophth. Cong.* **2**:624, 1929.

By February 29 the patient had made a good recovery; there were no headaches or vomiting.

Beginning on April 9 sixteen irradiation treatments were carried out before the patient was discharged from the hospital.

Up to May 15 recovery had been steady. The margins of the right disk were still blurred.

On May 22 the patient was readmitted to the Toronto General Hospital and discharged on June 6. She had lost considerable weight and most of her hair had fallen out over the posterior three quarters of the scalp. She recovered considerably with rest in bed; there was no vomiting or headache.

On May 27, 1937, when I first saw the patient, vision was $6/18 + 1$ in the right eye; there was no perception of light in the left. The pupil in the right eye was active, but the left was fixed and irregular, the iris being bound down by a complete posterior synechia. There was some ectropion of the uveal pigment. There was a dense opacity in the lens, and no fundus reflex could be seen. The right eye showed clear media, the borders of the disk were normal and there was slight pulsation in the vein and a few flecks of pigment in the foveola. The tension in the left eye with the Schiötz tonometer was over 80 mm. Slit lamp examination of the left eye showed a cloudy cornea, a shallow anterior chamber and vessels close to the posterior surface of the lens. Transillumination of the left eye seemed to indicate that a tumor was present, up and in. Enucleation was advised, but in the hope of being able to save the eye the patient was admitted to the hospital on March 30 for extraction of a cataract with a full iridectomy. The eye remained irritable, and the patient returned on May 30. At this time there was marked pain, pericorneal injection and a drawn pupil. Enucleation was again advised, and on May 31 the left eye was removed, local anesthesia being employed. The patient was discharged from the hospital on June 4. The pathologic report showed secondary glaucoma, secondary cataract, detachment of the retina and calcification of the choroid.

Further examination of this specimen was undertaken only after the sister's condition was verified on May 6, 1938.

Pathologic Examination of Right Eye (figs. 1 and 2).—The corneal epithelium was intact. The cornea was vascularized beyond the limbus. The anterior chamber showed complete obstruction of the filtration angles, and even in the central region the iris was firmly bound to the cornea. The ciliary bodies showed a moderate amount of free hemorrhage. The retina was extensively detached in the posterior part, and the choroid showed large deposits of pigment and exudate in which calcium salts had been deposited. On one side near the optic nerve there was an extensive mass of acellular exudate containing clumps of pigment. Heavy masses of calcium were seen close by and also numerous clefts, where apparently cholesterol crystals had been dissolved out in the process of preparation. Much partly absorbed hemorrhage was seen at the ora serrata, invaded by a light connective tissue. A small tumor, measuring 2.5 by 3 mm., was seen in the anterior portion of the retina. Large vessels were present at the side of the tumor. The tumor itself showed many fine vessels lined with a single layer of endothelium and numerous partly formed vessels. Special stains, iron hematoxylin and van Gieson's stain, showed the connective tissue surrounding the larger vessels. The main mass of tumor contained little connective tissue but numerous fine capillaries. In places clumps of endothelial cells suggested the process of formation of new vessels. There was a moderate amount of general mononuclear infiltration surrounding the area of the tumor and of light connective tissue that extended forward to the ciliary

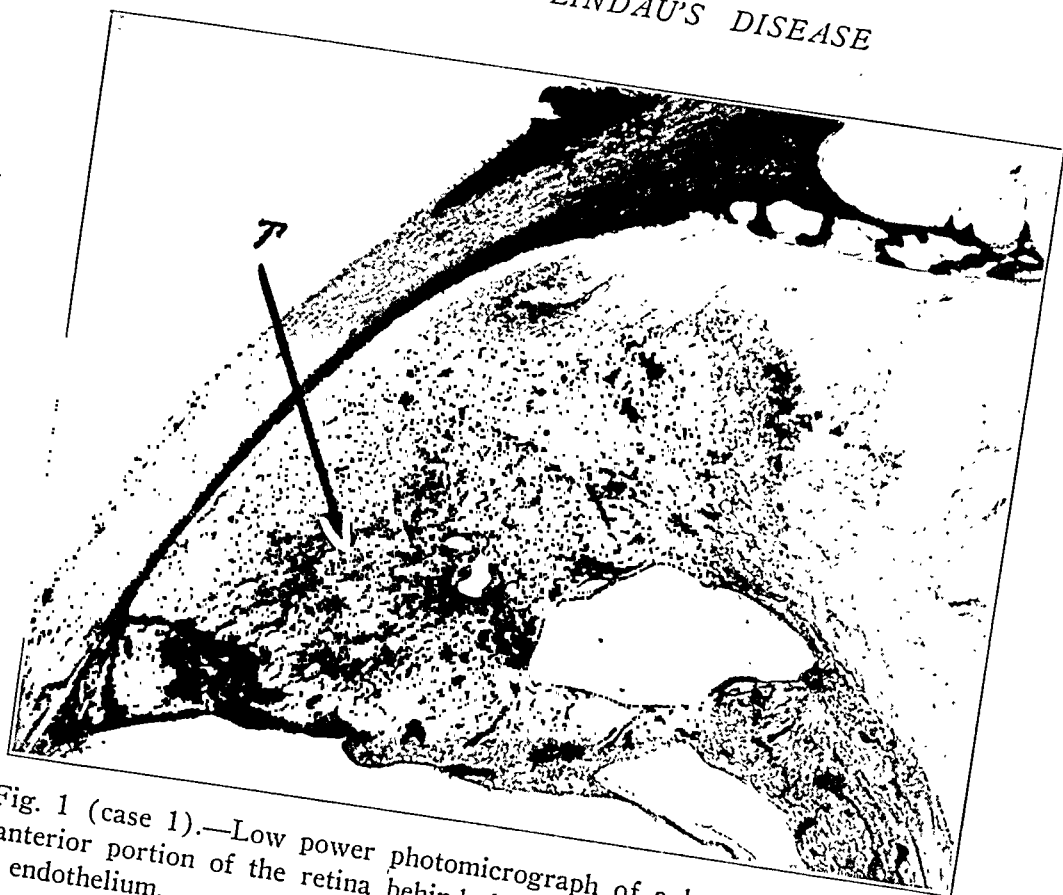


Fig. 1 (case 1).—Low power photomicrograph of a hemangioendothelioma in the anterior portion of the retina behind the ciliary body. The cysts were lined with endothelium.

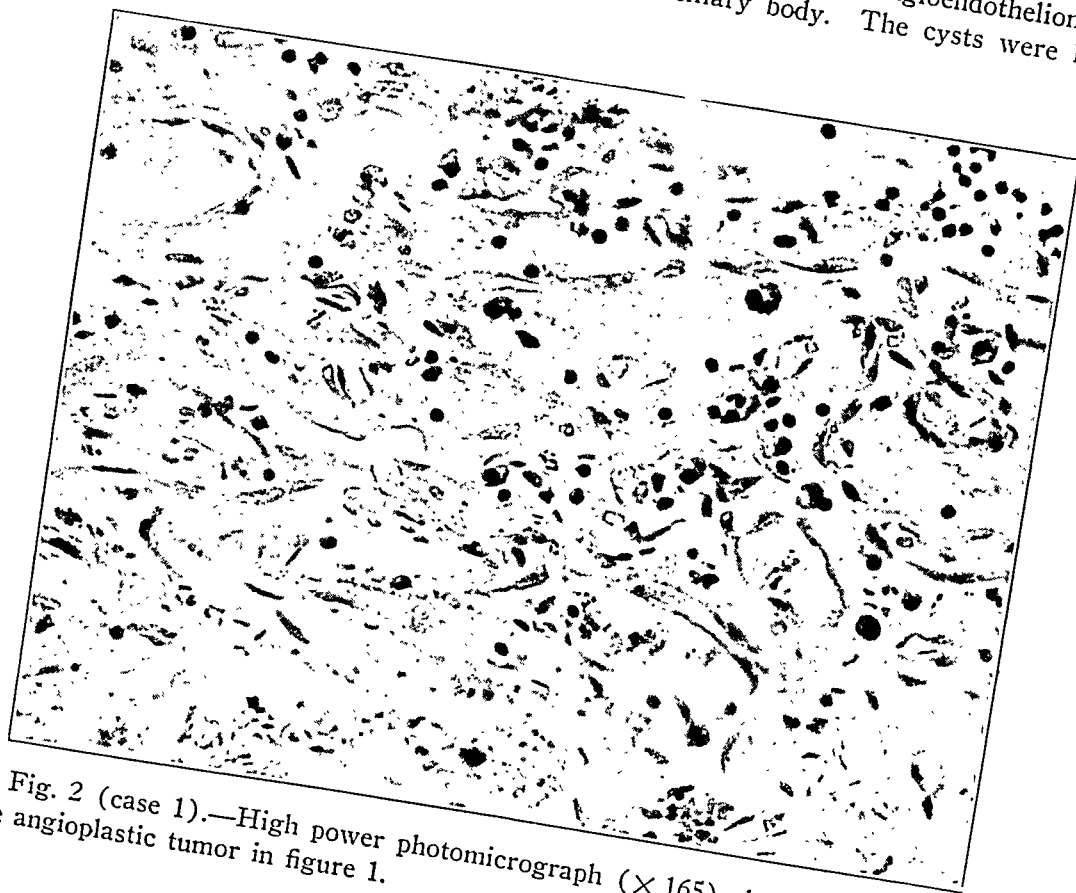


Fig. 2 (case 1).—High power photomicrograph ($\times 165$) showing the detail of the angioplastic tumor in figure 1.

bodies, which were not detached. Large cysts lined by endothelium were seen close to the tumor in the anterior portion of the retina.

A diagnosis was made of retinal hemangioendothelioma.

Additional Data.—On Aug. 7, 1939 the patient was readmitted to the hospital because of vomiting and headaches and inability to work. On August 10 a cerebellar tumor on the left side was removed by Dr. McKenzie, with difficulty on account of large vessels. On August 26 the patient was discharged after a good recovery. On November 8 histologic examination of the tumor showed small endothelium-lined spaces containing blood. These spaces were separated by proliferation of endothelial cells. The tumor presented the typical appearance of a hemangioendothelioma.

CASE 2.—Mrs. L. C. S., aged 29, a sister of the patient in case 1, consulted me on May 30, 1937. She had noticed floaters in the right eye for three years and had worn glasses for one year. No headaches had been experienced, but the eyes tired easily, and she had noticed loss of sight in the right eye. The vision in the right eye was 6/60 and in the left eye 6/9. The pupils were equal and showed direct light, consensual and convergence reflexes. Examination without a mydriatic showed in the right eye a large opacity of the vitreous with a strand attached to it. The fundus showed numerous irregular whitish flecks, which coalesced about the foveola. They were widely scattered, but decreased in size in the region of the upper and lower temporal vessels. The veins were full and markedly large, and there was a pulsation in the vein at the disk. The flecks were considered to be due to degenerative changes. The fundus of the left eye showed clear media and a normal cup; a slight temporal crescent was present with an occasional fleck near the disk; the foveola was clear. The vision of the right eye did not improve with glasses, but that of the left eye improved to normal. The patient was referred to her physician for a thorough physical examination.

On October 25 she complained of loss of vision in the right eye and stated that she had noticed occasional blurring in the left eye. The vision in the right eye was limited to ability to count fingers at 1 meter; that in the left eye was normal with correction. Examination of the fundus revealed only a slight increase of the condition previously described about the foveola. The patient was referred to Dr. McKenzie, as she staggered at times and stated that no libido was present.

On March 4 she was admitted to the Toronto General Hospital to the service of Dr. McKenzie. She stated that she had been well until October 1937, when she had severe frontal headaches, loss of vision in the right eye and vomiting. She had consulted Dr. McKenzie on March 2, but at that time Dr. McKenzie did not feel that any surgical procedure was necessary, and she had returned home.

On May 6 the field of the right eye showed a cut to 40 degrees below the nasal meridian. The fundus of the left eye showed blurred borders. A small hemorrhage was seen in the left eye well out at the periphery, near a small globular whitish mass. There was no history of past illnesses. The patient weighed 112 pounds (50.8 Kg.), having lost 23 pounds (10.4 Kg.) during the previous two months. Physical examination gave essentially negative results. Neurologic examination showed the third to the twelfth nerves to be normal. Examination of the eyes gave essentially the same results as on May 30. With the Romberg test the patient had a tendency to fall to the right, and she staggered to the right on walking. There was no paralysis or wasting.

A provisional diagnosis of cerebellar tumor on the right side, probably Lindau's disease, was made.

On May 16 Dr. McKenzie did a cerebellar exposure and found in the upper part of the left hemisphere a vascular lesion attached to the dura, lateral sinus and tentorium by a large, thin-walled vessel. Hemorrhage was free, and a tumor the size of a large walnut was removed. It shrank considerably on fixation and showed a yellow cyst, $\frac{3}{4}$ inch (1.9 cm.) in diameter, filled with yellowish fluid and lined by a pinkish smooth surface (fig. 3).

Pathologic Examination.—Tissue from the part of the tumor which is marked with a cross in figure 3 was examined and a diagnosis of hemangioendothelioma of the cerebellum made. The tumor tissue consisted of small vascular spaces separated by masses of endothelial cells, which were foamy in appearance. The silver stain showed a well marked network of reticulum fibers.

Ophthalmoscopic Examination.—On May 17 the right eye was dilated. There was a large floating mass well forward on the vitreous; above this was a strand that increased in thickness as it passed up and out to a mass at the periphery.

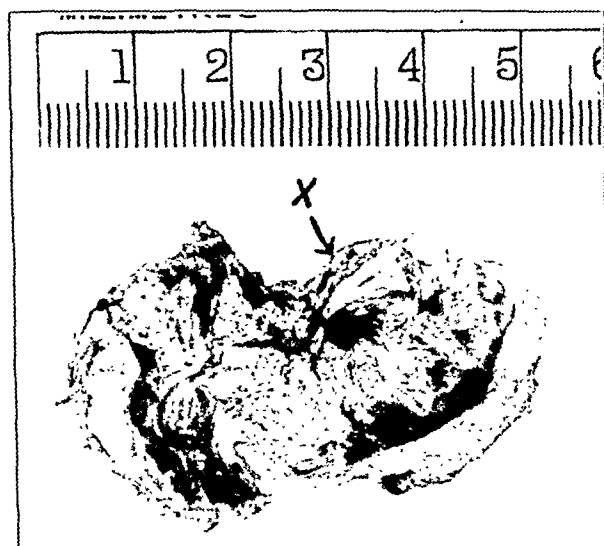


Fig. 3 (case 2).—Gross specimen of a cerebellar hemangioendothelioma. The section in figure 4 is from the area indicated by the cross.

With a + 12.00 sphere vessels were seen on this mass. A + 15.00 sphere showed only white connective tissue; some pigment was seen near the entrance of the greatly dilated vein and the artery. There was a veil of connective tissue over the disk, which ended in a knob seen with a + 5.00 sphere. The borders of the disk were blurred, especially above, below and nasally. On the temporal side heavy yellowish white areas with brilliant flecks were seen extending from the upper temporal to the lower temporal vessels; they were dense in the foveolar area. Patches of these multiple flecks decreased in number to the temporal side, and finally only faint discrete flecks were seen. Fine pigment (blood staining possibly) was seen along the lower temporal vessels, well out. The upper temporal artery was irregular, and where it passed over a white area the sharp borders presented slight bulgings, so that the caliber of the vessel was variable. This was more evident toward the periphery. The upper temporal vessels converged and passed into the aforementioned mass, which looked white in the central part and rather grayish at the edge. One small dilated capillary, or light hemorrhage, was seen at the lower border.

The media of the left eye were clear; the borders of the disk were slightly blurred above, below and on the nasal side, and the veins were dilated. There was one small flame-shaped hemorrhage below at the border of the disk and another small one above. There was a marked pulsation in the vein, while there was only slight pulsation in the right eye. Outside of an irregular light reflex, the foveolar region showed no change.

Course.—On May 20, 1939, the patient complained of further loss of vision. Her general condition was good. The fundus of the right eye showed a flat detachment near the tumor, with dark vessels, so that the artery and vein seemed alike unless followed from the disk. The central exudate and also the number of brilliant flecks had increased. The beading was more marked. Four 0.7 milli-curie radon seeds were inserted opposite the tumor, to give a total dose of 2.8

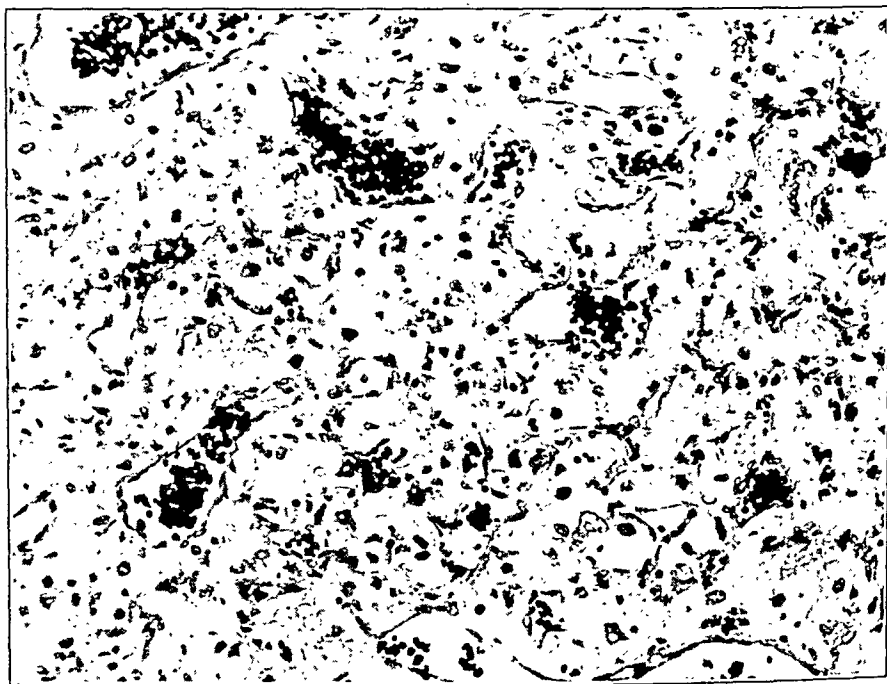


Fig. 4 (case 2).—High power photomicrograph ($\times 85$) showing the detail of the angioplastic cerebellar tumor.

millicuries for seven days. The removal was facilitated by dipping the seeds tied to a silk suture in liquid pyroxylin before insertion.

CASE 3.—Mrs. John R., the mother of the patients in cases 1 and 2, consulted Dr. Minnes, of Ottawa, Canada, in 1912, at the age of 22, according to her daughters, from whom an incomplete history was obtained. Dr. Minnes' notes, supplied by Dr. Wilbur Fraser, of Ottawa, stated that on July 2, 1912, the right eye had been sore for eleven years, following measles. It had been frequently painful, and this severe attack had lasted for one week, but sight had been wanting for over five years. A complete annular synechia was present, with opacities in the lens, and there was no perception of light. On July 3, 1912, enucleation was performed, and when the globe was opened it was found that there was complete ossification of the choroid, so that a bony cup was formed. A search was made

by Dr. Hooper in Dr. Webster's notes and also in the records of the Civic Hospital, but no report of an operation on the brain could be found, although the daughters stated that their mother died at the age of 32 after an operation on the brain for a suspected tumor.

Family P.—CASE 4.—Albert P., a white man aged 30, a gardener, was admitted to the Toronto General Hospital on Oct. 19, 1937. His chief complaints were attacks of dizziness and nausea, occasionally accompanied by headache. He had felt well until fourteen weeks before the present illness, when he suffered from his first attack of dizziness and nausea. A second attack followed three days later. Severe headaches would start at any time, even at 3 a. m., and last the whole day or at times only a part day. Sight was blurred in the last three weeks before admission, especially in the right eye. The patient tended to stagger when walking. There were no past illnesses, and inquiry into the personal history elicited nothing of importance. The family history showed that the father had become blind at 35 and died from cerebral complications at the age of 40. One sister had become almost blind during the past few months. One brother was alive and well.

Functional examination gave essentially negative results. The patient's memory was good.

Physical examination gave essentially negative results. The reflexes were hyperactive but equal throughout.

A differential diagnosis of an intracranial tumor or neurosis was made, with a provisional diagnosis of intracerebellar tumor on the right side.

On November 3 there was a fine nystagmus, and the eyes tended to drift to the right. The ophthalmoscope showed a small recent hemorrhage near the disk at 2 o'clock, in the right eye. There was loss of physiologic cupping; engorgement of the vein was present, and the margins of the disk were blurred.

On November 5 cerebellar decompression was done by Dr. McKenzie with the patient under ether administered endotracheally. A cyst containing yellow fluid was encountered in the midline of the cerebellum. A pink tumor, about 1 cm. in diameter, lying on the deep wall of the cyst, was picked up with the sucker and disappeared with the contents of the cyst, so that no tissue was obtained for pathologic examination.

The patient made a fairly satisfactory recovery and was discharged on December 24. He was able to stand with his heels together and his eyes closed; some nystagmus was still present, but the vomiting that he had experienced before the operation, especially if he turned on his back or to the right side, had disappeared.

By Jan. 21, 1938, the patient was greatly improved; there was still some dizziness, but the nystagmus was less.

By February 25 the patient was not able to read for any length of time, and he became dizzy with any exertion.

By July 17 he had returned to his former occupation, free from headaches and staggering.

Laboratory examination showed a negative Wassermann reaction, a cerebrospinal fluid pressure of 230 mm. and a blood pressure of 105 systolic and 75 diastolic. Otherwise the results of the examination were negative.

CASE 5.—On Dec. 28, 1938, Dr. Frank Turnbull, of Vancouver, B. C., wrote to Dr. McKenzie, stating that he had operated on Mrs. C. T., aged 29, a sister of the patient in case 4, six months previously for a cerebellar tumor of the hemangiomas cystic type. He stated that she had been totally blind for several months with bilateral retinal detachment. Dr. Turvey suggested that the condition

was Lindau's disease. The patient made an excellent recovery after the operation and resumed her occupation at the Institute for the Blind. It was confirmed that the blind father had suffered from severe headaches and died in 1916.

CASE 6.—The father of the patients in cases 4 and 5 became blind at 35 years of age. He died at about 40 years of age from complications of an intracranial nature. No record has been found for him.

COMMENT

It will be seen that in two generations of 2 families 6 cases of Lindau's disease have been found among 11 members. Of the remaining 5, only 1, Mrs. D., a sister of Miss R. and Mrs. S., was seen on July 16, 1938, at the age of 28. She consulted me as to whether a tinted +0.50 sphere which she had been using for each eye was necessary. Examination showed vision of 6/6—2 in each eye. The pupils were large and equal and showed direct light, consensual and convergence reflexes. The bulbar conjunctiva of the left eye showed a diffuse nevus 3 by 3 mm. near the limbus at 9 o'clock; two large vessels passed from this area to below the caruncle. The fields of the right and left eyes were full, and the patient recognized 1 mm. red, blue and green test objects with each eye. The fundi showed clear media and a pulsation in the vein on the disk, more in the left than in the right eye. In the left eye slight tortuosity of the vessels was noted. With correction of a +0.50 cylinder in each eye at 90 degrees vision was normal. A drop of an astringent solution was ordered for the slight redness that followed reading.

Mr. R., brother of the patients in cases 1 and 2, a school teacher, was not examined. His vision was said to be normal and his health good.

In family P. it was said that the unexamined brother had normal sight. The 2 unaffected members of the first generation, the father in family R. and the mother in family P. were said to have had normal sight.

CONCLUSIONS

1. The successful treatment of Lindau's disease seems possible, but further observation of treated patients is needed to show if the apparent relief of symptoms is lasting.

2. The ocular tumor may easily be missed at pathologic, as well as at clinical, examination. It was a small peripheral notch in the nasal field of 1 of my patients that led to the discovery of the tumor.

3. The possibility of angiomatosis retinae as a cause for blindness in the second and third decades must be borne in mind, for secondary glaucoma or hemorrhage may obscure the diagnosis.

4. Angiomatosis retinae occurs in the cerebral layer of the retina and may be one of multiple hemangioendotheliomas of the central nervous system.

5. Hemangioendothelioma is susceptible to treatment by irradiation.

6. Multiple hemangioendotheliomas must be demonstrated to justify a diagnosis of Lindau's disease, or if only one tumor is found pathologically, familial incidence must be shown.

7. A sister of the patients in cases 1 and 2 had a conjunctival nevus.

8. Both terms, Lindau and Hippel disease, must be retained.

DISCUSSION

DR. ALLEN GREENWOOD, Boston: I wish to mention a case which had been extremely interesting to me, that of a schoolteacher of 28, whom I presented at a meeting of the New England Ophthalmological Society some twelve years ago. I presented her because her fundus was easily seen and had two enormous veins in it, one running straight up and one running straight down. They were three or four times the size of the normal vein, and each one ended in a clear, round, shining cyst; a diagnosis of hemangioma was made. The patient, by the way, had a cousin who had had the same condition. The patient had lost her other eye because of separation of the retina and glaucoma. The eye was removed by Dr. Standish, and no examination was made, as far as I can find out. I sent the patient immediately to Dr. Harvey Cushing at the Peter Bent Brigham Hospital with the diagnosis I had made, to see if he could find any evidence of involvement of the cerebellum. He found none, but he had his artist make some beautiful pictures of the two cysts, which later became obstructed by disturbance in the vitreous. Dr. Cushing had roentgen irradiation used to see if it would stop the progress of the cysts and prevent blindness. A few years later the patient was led into my office by her uncle; she was led in because she was so dizzy she could not walk properly. She was sent again to the Peter Bent Brigham Hospital. Dr. Cushing had left, but Dr. Cutler made a diagnosis of cyst of the cerebellum, which he removed, and the last I knew of the patient she was living but with very poor vision. The cousin had been blind in both eyes, due to what was probably the same condition. I have not heard from this patient for several years, but, as I say, the last I knew she was still living, having had a tumor removed from the cerebellum. This was unquestionably a case of so-called Lindau's disease.

DR. WALTER S. ATKINSON, Watertown, N. Y.: Dr. MacDonald has referred to a case of Lindau's disease that I reported in 1932, and I wish to have recorded that this patient is still alive and well, although his gait is a bit unsteady. He has had two recurrences of the cerebellar cystic angiomatous tumor and has had two more operations, three in all. The eye with the angiomatosis retinae is blind due to total detachment of the retina. The other eye is not affected, and the vision is good.

DR. HENRY P. WAGENER, Rochester, Minn.: As MacDonald has stated, the diagnosis of a complete Lindau syndrome cannot be made

before the patient comes to necropsy, because one cannot demonstrate the presence of the other congenital anomalies which Lindau made a part of his complete syndrome. However, I do feel that one is justified in making a diagnosis of Lindau's disease when there is present a definite angiomatosis of the retina in association with a surgically proved hemangioma of the cerebellum. Craig, Kernohan and myself are reporting at the American Neurological Society 4 cases, in 1 of which necropsy was performed and the complete syndrome was confirmed, the presence of the angioma in the retina in each eye being confirmed histologically, and in the 3 of which confirmation was made surgically and the patients are still alive so far as we know.

SYNDROME OF NEUROMYELITIS OPTICA

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AND

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So complex is the nervous system and so manifold its functions that one and the same etiologic agent may produce many different syndromes by the pure chance of the site and the tempo of involvement. A disease affecting the upper or lower lobe of the lung, some portion of the liver or the right or left kidney may give similar symptoms and is considered under one designation. However, if there is a virus infection in the nervous system and it happens to attack the second cranial nerve, it may be called "papillitis"; if the vestibular portion of the eighth nerve is involved, one reads of "epidemic vertigo"; if the seventh nerve is invaded, there is facial paralysis.

As the possible combinations of involvement of the brain, cranial nerves, brain stem and spinal cord are numerous, so too are the designations or the syndromes.¹ These syndromes have a certain historical significance. They constitute fine descriptions of disease processes, but there is a tendency to confusion and an impression that many distinct and widely different disease processes are being dealt with. For instance, one reads in the French literature such terms as neuritis, neuronitis, plexitis, schwannitis, ganglionitis and many combinations involving the spinal cord.

Such titles are useful as clinical guides if it is understood that each may represent merely a phase or subdivision of a major group and not a distinct disease.

In the past there has been a tendency to subdivide the entire group of nonsuppurative infections of the nervous system into four major concepts. According to Brain² and Berliner,³ these include multiple sclerosis, Schilder's disease, acute disseminated encephalomy-

1. Strauss, I.; Rabiner, A. M., and Ginsburg, S. W.: The Clinical, Bacteriological and Epidemiological Aspects of Encephalomyelitis, *A. Research Nerv. & Ment. Dis.*, Proc. **12**:262, 1932. This article contains different points of view on this subject and an extensive bibliography.

2. Brain, W. R.: Critical Review: Disseminated Sclerosis, *Quart. J. Med.* **23**:343, 1930.

3. Berliner, M. L.: Acute Optic Neuritis in Demyelinating Diseases of the Nervous System, *Arch. Ophth.* **13**:83 (Jan.) 1935.

elitis and neuromyelitis optica. Even this subdivision is open to criticism, because there are many neurologists who believe that these designations apply to the same disease in its different manifestations. There are many able neuropathologists, among them Hassin,⁴ who make a fundamental distinction between these various disease processes.

There is need for a unified terminology, which it is hoped will be forthcoming when the etiologic agent can be better identified. At the present time, the majority of authors agree with Rivers⁵ that the cause of such illnesses is a virus and that the clinical course of the disease is subject to marked variations.

Among the combinations of areas affected, which may turn up with remarkable frequency (like 7 and 11 on dice), are the optic nerve and the spinal cord. This combination is well known to every ophthalmologist and to neurologists, who, if they encounter a case of retrobulbar optic neuritis, look for and expect to find evidence of pathologic changes in the spinal cord at the same time or at a later date. When the involvement of the combined areas is acute and striking, it has been called neuromyelitis optica. The earlier case reports which called attention to this association were those published by Albutt⁶ in 1870 and by Erb in 1879.⁷ Devic⁸ in 1894 laid stress on this syndrome by means of case reports and pathologic studies. Important papers also were contributed by Goulden⁹ in 1914 and by Beck¹⁰ in 1927. Because of Devic's important contribution, this syndrome has been referred to as Devic's disease; it has also been called neuromyelitis optica or neuro-optica myelitis.

It is with a certain hesitancy that the following case report is offered. However, it is hoped that it may prove interesting not only by itself but as a basis from which several of the problems of this disease may be discussed.

4. Hassin, G. B.: *Histo-Pathology of the Peripheral and Central Nervous Systems*, Philadelphia, W. B. Saunders Company, 1934.

5. Rivers, T. M.: *Relation of Filtrable Viruses to Diseases of the Nervous System*, *Arch. Neurol. & Psychiat.* **28**:757 (Oct.) 1932.

6. Albutt, T. C.: *On the Ophthalmoscopic Signs of Spinal Disease*, *Lancet* **1**: 76, 1870.

7. Erb, W.: *Ueber das Zusammenvorkommen von Neuritis optica und Myelitis subacuta*, *Arch. f. Psychiat.* **10**:146, 1879.

8. Devic, M. E.: *Myélite aiguë dorso-lombaire avec névrite optique*, *Cong. franç. de méd.* **1**:434, 1894.

9. Goulden, C.: *Optic Neuritis and Myelitis*, *Tr. Ophth. Soc. U. Kingdom* **34**:229, 1914.

10. Beck, G. M.: *A Case of Diffuse Myelitis Associated with Optic Neuritis*, *Brain* **50**:687, 1927.

REPORT OF A CASE

L. C., an intelligent and previously healthy Italian girl of 17, was admitted to the Lakeside Hospital, to the service of Dr. J. T. Wearn, on March 12, 1937. She was referred because of intense pains in the lower part of the back and legs, of five days' duration, and of increasing dimness in vision. Three weeks prior to her admission she had contracted a "cold" and had symptoms of coryza. This was followed by a backache, malaise and dizziness. One week later she had a temperature of 103 F. accompanied by severe pains radiating down the legs. Three days before entering the hospital she was troubled by pain in the right eye and gradual loss of vision. In the succeeding days she experienced difficulty in voiding.

The initial examination revealed a tall girl of 17 in a good state of nutrition, lying restlessly, with a temperature of 100.4 F., a pulse rate of 100 and a respiratory rate of 20. There was no evidence of infection of the respiratory tract, and the significant features were restricted to the neurologic study. The right eye was completely blind and the vision in the left eye appreciably diminished, particularly for colors. There were occasional spasmodic contractions which produced odd movements of the eyeballs; there was slight weakness of convergence. The right pupil was dilated and failed to respond to light, but the reactions of the left pupil were normal.

Examination of the right fundus revealed slight elevation of the nerve head, with blurring of the margins, and exudate and hemorrhages on its surface. The veins were engorged and tortuous, but the arteries were of average caliber. The left optic disk showed only slight haziness of its borders and moderate fulness of the veins. All four extremities were weak, and the tendon reflexes were hyperactive. The neck was slightly rigid, and the Kernig sign was positive. Light touch revealed hyperesthesia over the lower extremities. There was urinary retention. The laboratory findings as regards the spinal fluid were pathologic. The initial pressure was 250 mm. of water. The fluid was clear; the Pandy reaction, 2 plus; the lymphocyte count, 26; the protein content, 138 mg. per hundred cubic centimeters; the sugar content, 69 mg., and the colloidal gold curve, 2210.

The urine was normal, the white cell count being 11,400, with 74 per cent polymorphonuclears, 16 per cent lymphocytes and 8 per cent monocytes. Cultures of the blood were negative; cultures of the spinal fluid and inoculation of laboratory animals by Dr. Parker did not reveal a causative organism or virus.

The clinical course was at first progressively downhill. Within several days the vision in the left eye became more dim, until the patient was totally blind in both eyes. Both optic disks were elevated, about 3 diopters in each eye. The left nerve head was definitely blurred, with multiple hemorrhages and exudate.

Spinal fluid pressure at this time was 270 mm. of water. The Pandy reaction was 4 plus; the protein content, 191 mg. per hundred cubic centimeters, and the cell count, 75 lymphocytes.

At about this time there were increasing pains in the legs, with almost complete paralysis on the right side and partial paralysis on the left. Mentally, the patient became irritable, sometimes confused.

The sensory tests revealed patchy areas of diminished sensation, particularly in the lower extremities. Later, there was involvement of the arms, with sensory diminution and motor weakness. The abdominal reflexes were absent.

After the height of the illness was reached, about March 25, there was gradual improvement. On March 28 visual perception returned in the left eye, and several days later the patient could detect light in the right eye. In the subsequent days the vision improved steadily, and the pathologic changes in the eyeballs gradually

disappeared. On April 11 the patient could move her right leg, and on April 17 the reflexes had returned. On April 21 the patient was able to void voluntarily.

The patient was discharged on June 1, after three and a half months of illness, considerably improved. The vision in the right eye had returned to 6/12 and was 6/6 in the left eye. Studies of the visual fields showed moderate constriction for color and form in the right eye, while the left eye was normal. The optic nerve head on the right side showed moderate pallor, while the appearance of the left disk was essentially normal. The return of motor function was practically complete. The sensory losses had likewise cleared up, save for a residual area of hyperesthesia over the area of the lateral cutaneous nerve of the right leg. The personality disturbances had cleared up, and the patient appeared to be friendly and well adjusted.

During the two year period which has followed hospitalization, we have had occasion to make several subsequent examinations. The vision has remained normal in the left eye and only slightly impaired in the right. The fields are now complete, both for form and for color. Only slight pallor of the right disk remains. The paresthesia of the right side is another mild residual symptom.

Summary.—The patient was a girl in the late teens, who suffered from acute bilateral optic neuritis plus diffuse myelitis. The blindness preceded the definite signs of involvement of the spinal cord, which was manifested by complete paralysis of both legs, loss of sphincter control and marked hypesthesia of the legs and thighs. In the course of several months, the patient regained completely the vision in the left eye and had only a minimal impairment in the right. There was an almost total return of both sensory and motor function.

COMMENT

Differential Diagnosis.—In the case reported an early diagnosis of disseminated involvement, before the changes in the spinal cord appeared, would have been difficult. Cases of marked bilateral optic neuritis present a serious problem in the differentiation of these changes due to inflammation of the nerve and those resulting from the increased intracranial pressure associated with tumor of the brain. Goulden distinguished between those cases in which the involvement was strictly retrobulbar, with the alteration in the appearance of the disk, and those in which a true papillitis was present, due to changes in the most distal segment of the nerve. The ophthalmoscopic picture of such a papillitis may be of varying degree, from simple hyperemia to marked swelling with several diopters' elevation.

This difficulty in diagnosis was emphasized by Schaltenbrand,¹¹ who stated that bilateral optic neuritis may frequently be mistaken for epi-

11. Schaltenbrand, G.: Hirngeschwulstähnliche Erkrankungen die reine Geschwülste sind, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **161**:162, 1938; abstracted, *Year Book of Eye, Ear, Nose and Throat*, Chicago, The Year Book Publishers, Inc., 1938.

dence of tumor of the brain. Walsh¹² called attention to 2 of 4 cases seen at the Johns Hopkins Hospital in which an erroneous diagnosis of tumor of the brain was made. Likewise, McKee and McNaughton¹³ reported cases of papillitis, in 1 of which exploratory craniotomy was done and in the other of which ventriculographic examination was made.

In cases of optic neuritis the extent to which the edema spreads into the surrounding retina may be of diagnostic aid, but there are many instances in which the criteria suggested by Leslie Paton¹⁴ may offer valuable assistance in excluding a lesion due to increased intracranial pressure. Paton stressed the fact that the loss of vision in optic neuritis precedes the ophthalmoscopic changes. The inference from this statement is that in the presence of edema of the optic nerve from papillitis blindness may be present, whereas ordinary papilledema precedes significant visual changes. This author added that in neuritis the changes in the fundi are of relatively short duration, the disks may show no trace of change after the swelling has subsided and the recovery of vision parallels the subsidence of swelling.

As to the danger of watchful waiting over a short period of time, to determine whether a questionable case of "choked disks" is due to optic neuritis, Gordon Holmes¹⁵ observed that papilledema is too frequently considered an immediate danger to vision. Holmes stated: ". . . in even the most experienced hands operation, whether radical or palliative, entails considerable risk of life, and for this reason as well as on other grounds, as the lack of symptoms that can establish a definite localization, delay is often advisable." In our case the problem of differential diagnosis was rendered more difficult by the moderate elevation in the spinal fluid pressure, 250 mm. of water.

Treatment.—Obviously, because of the unknown causation, there is no specific treatment available, and many measures have been tried, with little or no success (Fralick and DeJong¹⁶). In this instance we found that large doses of salicylates were effective in alleviating the deep-seated pain in the back and legs. We believe exhausting treatment, such as fever therapy, to be contraindicated. The serious complication of paralysis of the bladder was treated by drainage and

12. Walsh, F. B.: Neuromyelitis Optica, Bull. Johns Hopkins Hosp. **56**:183, 1935.

13. McKee, S. H., and McNaughton, F. L.: Neuromyelitis Optica, Am. J. Ophth. **21**:130, 1938. Woods, A. C., in discussion on McKee and McNaughton, pp. 136-137.

14. Paton, L.: Papilledema and Optic Neuritis, Arch. Ophth. **15**:1 (Jan.) 1936.

15. Holmes, G.: The Prognosis in Papilledema, Tr. Ophth. Soc. U. Kingdom **57**:3, 1937.

16. Fralick, F. B., and DeJong, R. N.: Neuromyelitis Optica, Am. J. Ophth. **20**:1119, 1937.

irrigation with a mild antiseptic. The method introduced by Monroe and Hahn¹⁷ was used. Nursing care was of course of tremendous value in the prevention of trophic ulcers and stasis.

Prognosis.—Many authors (Goulden, Rea¹⁸ and Fralick and DeJong) give a mortality rate of 50 per cent for neuromyelitis optica. Those persons who survive may either recover completely or manifest a residual paralysis or some degree of secondary atrophy of the optic nerve. Fralick and DeJong observed that the rapidity of onset was not a deciding factor in the prognosis.

As regards the vision, the literature mentions the fact that "white disks" may occasionally be seen in the presence of perfectly normal vision. Whether this is due simply to atrophy of the nerve fibers or in how far it represents a proliferation of glial tissue or perhaps a diminution in the number of small blood vessels is not known. As King¹⁹ has pointed out, Rönne would explain this retention of normal visual acuity in the presence of an apparent atrophy of the optic nerve as indicating the grossness of tests employing Snellen charts, which may permit registration of normal vision in the presence of the destruction of many of the nerve fibers.

Relation of Neuromyelitis Optica and Multiple Sclerosis.—Attention is directed particularly to retrobulbar neuritis, which is part of this syndrome. The incidence of unilateral retrobulbar neuritis as an early symptom of multiple sclerosis is well known. Adie²⁰ analyzed the figures for several London hospitals and concluded that in 38 per cent of cases this was the first symptom and in 58 per cent an early symptom. Lillie,²¹ of the Mayo Clinic, studied some 500 cases and reported that disturbed vision was the initial episode in 15 per cent and a second or third episode in 35 to 40 per cent. The long duration of the natural remissions in multiple sclerosis makes the follow-up study in these cases sometimes extremely difficult. Adie reported that forty-seven years was the longest interval on record between the initial visual disturbance and the later onset of disseminated symptoms of multiple sclerosis. He therefore suggested that acute retrobulbar neuritis may, in some

17. Monroe, D., and Hahn, J.: Tidal Drainage of the Urinary Bladder, New England J. Med. **212**:229, 1935.

18. Rea, R. L.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1938, pp. 324-326.

19. King, C., in discussion on Kiely, C. E.: Neuromyelitis, Ohio State M. J. **32**:741, 1936.

20. Adie, W. J.: Acute Retrobulbar Neuritis in Disseminated Sclerosis, Tr. Ophth. Soc. U. Kingdom **50**:262, 1930.

21. Lillie, W. J.: Clinical Significance of Retrobulbar and Optic Neuritis, Am. J. Ophth. **17**:110, 1934.

instances, be the only manifestation of this disorder. Therefore, in the present state of knowledge the ultimate prognosis must be guarded.

We have reviewed the cases of encephalomyelitis seen at the University Hospitals during the ten year period from 1927 to 1937, so as to gain some perspective of the ultimate course in some of these cases. Of 5 cases reported by one of us (J. L. F.²²) under the title of "Mild Migratory Myelitis," progressive symptoms characteristic of multiple sclerosis have developed in 3. Of 20 other cases in which the condition was considered encephalomyelitis, sequelae or later developments that could be considered multiple sclerosis have arisen in 5. Our opinion, therefore, concurs with that expressed by McAlpine,²³ who considered neuromyelitis optica to be but a syndrome of multiple sclerosis.

Although there is an apparent striking difference between neuromyelitis optica and chronic multiple sclerosis, the distinction may be merely that of the time element rather than an actual difference in etiologic agent or the pathologic process. In neuromyelitis optica the changes occur simultaneously or consecutively in a matter of several weeks, while in multiple sclerosis these same structures may be affected in the course of years. One may think of the analogy of this relation to various forms of tuberculosis; there may be galloping tuberculosis, with an acute pulmonic involvement, and chronic fibroid phthisis. In one instance the process may be acute and last a few weeks; in the other instance the disease process may extend over a period of many years, though the causation of the two conditions is the same.

CONCLUSION

We have presented briefly a case of neuromyelitis optica, which is interesting because of the marked changes in the vision and the eye-grounds, which subsequently disappeared almost entirely. The difficulties in distinguishing papillitis from papilledema are noted. We have used the designation of neuromyelitis optica with the understanding that this is but a descriptive designation for what is most likely but a form of multiple sclerosis.

22. Fetterman, J. L.: Mild Migratory Myelitis, *Ohio State M. J.* **32**:1090, 1936.

23. McAlpine, D.: Acute Disseminated Encephalomyelitis: Its Sequelae and Its Relationship to Disseminated Sclerosis, *Lancet* **1**:846, 1931.

VACCINIA WITH OCULAR INVOLVEMENT

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Many cases of vaccinia involving the lids and cornea have been reported, so that the reports of 2 additional cases of vaccinia of the lids, which are included in this article, are of comparatively little interest.

The purpose of this paper is to discuss measures that offer some promise of inhibiting the action of the vaccinia virus or that will protect the cornea against becoming involved.

The incidence of inoculation of the lids or cornea is apparently small when one considers the large number of vaccinations that are done each year. However, many cases are probably not reported, and possibly some may not be recognized.

When ocular involvement does occur, the lids are usually the first to become affected, and the cornea is probably inoculated later by the virus from the lesions on the lids or the conjunctiva. However, a number of cases have been reported in which there has been direct inoculation of the cornea.

It should be emphasized that because of an occasional careless inoculation of the eye after vaccination, one should not be influenced against vaccination. Evidently this excellent and simple method of preventing smallpox has not been taken advantage of as generally as it should be, for in 1938 the United States Public Health Service reported over 14,000 cases of smallpox in the United States. This should be of concern to the ophthalmologist when he recalls the serious impairment of vision suffered by many persons who were fortunate enough to recover from smallpox before the introduction of vaccination. No doubt there are still many eyes affected by this disease.

In 1919 Bedell¹ reported a case of vaccinia involving the lids and cornea, and his appended bibliography includes 92 cases with ocular involvement. In the same year Meder² reported a case in which the internal canthus and cornea were involved. In his bibliography there are 27 additional references.

1. Bedell, A. J.: *Tr. Am. Ophth. Soc.* **17**:273-281, 1919.

2. Meder, E.: *Veröffentl. a. d. Geb. d. Med.* **9**:405-434, 1919.

Ninety-one cases of vaccinia with ocular involvement were collected from the literature covering a period from 1919 to date.³ In 32 of these the cornea was affected. Therefore, some impairment of vision probably occurs in approximately one third of the cases reported.

3. Amshel, F.: *Vaccinia*, Arch. Dermat. & Syph. **28**:287 (Aug.) 1933. Ander-vont, H. B., and Friedenwald, J. S.: Bull. Johns Hopkins Hosp. **42**:1-7, 1928. Anelli, D.: Boll. d'ocul. **11**:1265-1268, 1932. Aron: Klin. Monatsbl. f. Augenh. **74**:233, 1935. Ball, J. M., and Toomey, N.: *Vaccinia of the Eyelids by Homo-Inoculation* J. A. M. A. **79**:935-936 (Sept. 16) 1922. Benetazzo, G.: Dermosifilografo **11**:169-175, 1936. von Berger, F.: Ann. di ottal. e clin. ocul. **51**:262-266, 1923. Blessig, E.: Eesti Arst **11**:435-436, 1932; abstracted, Zentralbl. f. d. ges. Ophth **28**:410-411, 1933. Busacca, A.: Lettura oftal. **2**:337-354, 1925. Camus, L., in discussion on Delord and Villard: Bull. Acad. de méd., Paris **97**:610-611, 1927. Colden: Klin. Monatsbl. f. Augenh. **74**:233, 1925. Cronstedt, L.: Upsala läkaref. förh. **31**:317-328, 1926; Lettura oftal. **8**:223-241, 1931. Espino, J. M.: Rev. cubana de oftal. **2**:253-256, 1920. Folk, M. L., and Taube, E. L.: Am. J. Ophth. **16**:36-39, 1933. Frei, W.: Zentralbl. f. Haut- u. Geschlechtskr. **39**:498, 1932. Friede, R.: Klin. Monatsbl. f. Augenh. **85**:427-430, 1930. Fuchs, A.: Wien. med. Wchnschr. **75**:2504-2506, 1925. Gabriélidès, A.: Des conjonctivites, Constantinople, Imprimerie "Estia," Galata, 1921. Gaté, J.; Michel, P.-J., and Boyer, C.-E.: Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Lyon) **39**:46-48, 1932. Grüter, W.: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **46**:414-416, 1927. Hambresin: Bull. et mém. Soc. franç. d'opht. **41**:267, 1929. Horay, G.: Orvosi hetil. **67**:296-297, 1923; abstracted, Zentralbl. f. Haut- u. Geschlechtskr. **11**:48, 1924. Jaensch: Klin. Monatsbl. f. Augenh. **74**:233, 1925. Jendralski, F.: ibid. **74**:233, 1935. Kerl, W.: Arch. f. Dermat. u. Syph. **148**:610-613, 1925. Lombardo, C.: Sperimentale, Arch. di biol. **75**:131-144, 1921. Ludwig, A.: Arch. f. Augenh. **109**:346-352, 1935. Marín Amat, M.: Bull. et mém. Soc. franç. d'opht. **49**:85-90, 1936; Arch. de oftal. hispano-am. **32**:427-432, 1932; abstracted, Med. ibera **1**:73-74, 1932. Meyers: Ztschr. f. Med. beamte **35**:494-497, 1922. Montanelli, G.: Lettura oftal. **8**:135-142, 145-150, 153-158 and 161-165, 1931. Morax, V.: Précis d'ophtalmologie, ed. 4, Paris, Masson & Cie, 1931, pp. 215-217. Motolese, F.: Lettura oftal. **7**:336-338 and 341-343, 1930. Munns, G. F.: Am. J. Ophth. **14**:1037-1039, 1931. Nunn, L. L.: Northwest Med. **30**:194, 1931. Onfray, R.: Bull. et mém. Soc. franç. d'opht. **42**:267-268, 1929. Ortmann, K. K.: Hospitalstid. **75**:285-296, 1932. Pacelli: Not. di diagnost. e terap., 1929, no. 6. Portoghese, G.: Ann. di ottal. e clin. ocul. **62**:308-320, 1934. Sanguinetti, C.: Gior. di ocul. **3**:157, 1922. Schmelzer, H.: Ztschr. f. Augenh. **80**:356-359, 1933. Sédan, J.: Ann. d'ocul. **159**:604-610, 1922. Sexe: Bull. et mém. Soc. franç. d'opht. **42**:262-269, 1929. Sezer, F. N.: Accidental Vaccination of Conjunctiva, Arch. Ophth. **20**:89-91 (July) 1938. Simidu, S.: Acta Soc. ophth. japon. **41**:167-172, 1937. Sinaiko, A. A.: Keratitis Postvaccinalosa, Arch. Ophth. **5**:91-92 (Jan.) 1931. Stark, A. M.: Arch. f. Dermat. u. Syph. **170**:38-60, 1934. Tessier, G.: Lettura oftal. **6**:137-142, 1929. Thies: Klin. Monatsbl. f. Augenh. **74**:227-228, 1925. Toomey, N.: Am. J. Ophth. **5**:292, 1922. Venturi, T.: Dermosifilografo **3**:576-582, 585-588, 591-594 and 597-601, 1928. Vita, A.: Atti d. cong. d'oftal. (1925), 1926, pp. 279-280. Wirtz, K.: Thesis, Cologne, no. 3, 1928, pp. 13-14 and 21-22. Young, C. A.: Virginia M. Monthly **52**:171-172, 1925.

REPORT OF CASES

The vaccinia of the lids in the following 2 cases occurred in children after the usual vaccination for smallpox.

CASE 1.—D. L., a girl aged 5 years, had an ocular lesion which was diagnosed as a sty by her physician. The unusual appearance of the lesion prompted a more careful inquiry into the history, and the following information was obtained from the mother.

Eleven days before the lesion on the lid appeared, the child had been vaccinated. Four days later the usual papule developed. Two days later a small lesion developed on the right lower lid, which gradually became yellowish. The lids became very red and swollen, so that the child was unable to open her eye. When she was first seen five days after the appearance of the lesion on the lid, examination showed the following picture:

The lids were red and swollen, and there was an ulcer 8 mm. long on the margin of the lid near the outer canthus. The surface of the ulcer was covered with a yellowish, soft, moist coating. Just nasal to this ulcer was another smaller one covered with a grayish coating. On the upper lid, in a corresponding position, were three small ulcers of similar appearance. The lids were indurated in the vicinity of the ulcers. There was no fluctuation, and when the scabs were removed a little serous fluid exuded but no pus. A smear and culture showed no specific organism. There were marked tenderness and swelling of the preauricular and submaxillary glands and some conjunctival secretion. The cornea was not affected. In two weeks the ulcers had healed, leaving slightly depressed scars with the loss of a few cilia.

CASE 2.—J. P., a girl aged 3½ years, was vaccinated (on the right thigh) eight days prior to the first visit. Three days after the vaccination a small red spot was noticed on the margin of the left lower lid, which gradually increased in size. Four days later the mother said that it developed into a sty and that the next day a similar lesion appeared on the upper lid, opposite the first.

The history of vaccination was not obtained until the nature of the lesion had been observed.

The appearance of the ulcers and induration of the lids was as described in case 1, with swelling and tenderness of the preauricular and submaxillary glands. Several more small ulcers developed on either side of the original ulcers along the margins of the lids and two small ones developed on the skin of the lower lid. All were covered with a moist gray coating. Five days later, or ten days after the first lesion appeared, the ulcers had healed, leaving slightly depressed scars and loss of cilia. The cornea was not affected.

The treatment was symptomatic, consisting of frequent gentle irrigations with boric acid solution, the application to the lids of an ointment containing mercury bichloride and the use of ice compresses. Trauma was particularly avoided.

During the two or three days when the lids were greatly swollen and difficult to open, no force or lid retractors were used because of the danger of inoculating the cornea. Cuffs were used on the child's arms to prevent her bending the elbows, so that she could not rub the eye.

The pustules on the lids were not as tender or painful, as is usually the case with a hordeolum.

COMMENT

Fortunately, the pustule and resultant ulcer on the lids is not followed by as much scarring as after the usual vaccination. Possibly this is due to the fact that there is less virus present and that it is continually kept moist by the tears and conjunctival secretion. Also, some immunity may develop from the original vaccination.

In cases reported in which the cornea became involved, keratitis resembling keratitis disciformis developed. It was usually centrally located and often accompanied by serous iritis.

The clinical picture of vaccinia involving the lids is characteristic; so with a history of recent vaccination or of the patient coming in contact with the virus, the diagnosis can be made with reasonable certainty. However, in case of doubt the laboratory tests can be of assistance.

Tanaka ⁴ first observed that a suspension of vaccine lymph undergoes flocculation when brought in contact with immune serum. He also observed that fluid from a serous effusion of lesions of smallpox which is left in contact with a suspension of vaccine lymph for several days at 37 C. produced a coagulation phenomenon akin to the Pfeiffer-Gruber-Widal reaction. These observations have been confirmed by others.

For the diagnosis of vaccinia or variola, both the complement fixation and the agglutination test may be used. Gordon ⁵ recommended doing both at the same time. The result of the complement fixation test is obtained on the first day and may be checked by the agglutination test on the following day. Both reactions appear to be specific for vaccinia and variola.

In the report of Gordon ⁵ and that of Craigie and Tulloch,⁶ a description of the detailed technic of the tests is given.

A simple test for vaccinia is to inoculate the ear of a white rabbit with the vaccinal material. On the second day a red palpable swelling occurs. Increased vascularization is noted readily on looking through the ear when it is held up to the light. On the fourth day the site of inoculation appears as a firm, yellowish, elevated area. It soon becomes umbilicated, dries up, with scab formation, and disappears by the twelfth day.

4. Tanaka: *Centralbl. f. Bakt. (Abt. 1)* **32**:726, 1902.

5. Gordon, M. H.: *Studies of the Viruses of Vaccinia and Variola*, Medical Research Council, Special Report Series, no. 98, London, His Majesty's Stationery Office, 1925.

6. Craigie, J., and Tulloch, W. J.: *Further Investigations on the Vaccinia-Variola Flocculation Reaction*, Medical Research Council, Special Report Series, no. 156, London, His Majesty's Stationery Office, 1931.

The obvious objection to this test is that it requires more time than the complement fixation and agglutination tests. However, antivaccinial serum necessary for the last-mentioned tests may not be available, and at least eight days are necessary for its preparation.

Inoculation of the rabbit's cornea is not satisfactory. According to Defries and McKinnon,⁷ a positive take on the cornea occurs in only about 50 per cent of the rabbits. Fortunately, the cornea seems resistant to the virus.

With the definite diagnosis of vaccinia of the lids, one's chief concern is to try to prevent corneal involvement.

In 1896 Sternberg⁸ observed that the serum of an animal rendered immune to vaccinia possesses the capacity of destroying that virus when brought in contact with it in vitro. This observation has been confirmed by others, and it is also true in the human being. The virus neutralization and virus agglutination properties of antivaccinial serums suggest the local use of the serum to inactivate the action of the virus and prevent involvement of the cornea.

Béclère, Chambone and Ménard⁹ first reported a passive immunity in the fetus from the mother, which has been confirmed by others. Sato¹⁰ reported that young rabbits of vaccinated mother rabbits possessed corneal as well as cutaneous immunity.

Hlava and Honl¹¹ reported that the subcutaneous injection of the serum of immunized calves protected normal calves against subsequent inoculation with the vaccinia virus. Similar injections in school children of 0.6 to 1 cm. of immune serum per kilogram of body weight produced protection against subsequent inoculation with vaccinia. This has been confirmed by others.

Camus,¹² and Henseval and Convent¹³ pointed out that the preventive action of immune serum was greater than the curative action. By multiple vaccinations, Gordon⁵ was able to produce more immune serum, so that smaller doses could be used.

From these observations it seems logical to expect that some immunity would be produced by the injection of immune serum. If the serum could be injected as soon as the diagnosis of vaccinia of the lids is made, sufficient immunity might be produced to prevent corneal involvement.

7. Defries, R. D., and McKinnon, N. E.: *Am. J. Hyg.* **8**:107, 1928.

8. Sternberg: *Centralbl. f. Bakt. (Abt. 1)* **19**:857, 1896.

9. Béclère, A.; Chambone, and Ménard: *Ann. Inst. Pasteur* **13**:81, 1899.

10. Sato, K.: *Ann. Inst. Pasteur* **32**:481, 1921.

11. Hlava, J., and Honl, I.: *Wien. klin. Rundschau* **9**:625, 643, 1895.

12. Camus L.: *Compt. rend. Soc. de biol.* **73**:294, 1912.

13. Henseval, M., and Convent, A.: *Bull. Acad. roy. de méd. de Belgique* **26**: 251, 1912.

We were unable to demonstrate this satisfactorily with rabbits, because the cornea is apparently resistant to the vaccinia virus. However, sufficient immunity may be produced in the rabbit to prevent a take on the ear, while with rabbits not previously immunized a good take is practically always obtained.

The virus of vaccinia is made inactive by several drugs. Gordon⁵ found that potassium permanganate in dilutions up to 1:100,000 in contact with the virus for one hour at room temperature (17 C. [62.6 F.]) inactivated the virus. Also, mercuric chloride in a dilution up to 1:10,000 destroyed the virus.

Kligler and Bernkopf¹⁴ reported that relatively small amounts of ascorbic acid inactivated many times the infective dose of the vaccinia virus. A dilution of 1:2,000 inactivated one hundred times the infective dose of the virus when incubated for three hours. They suggested that the ascorbic acid acts as an oxidation-reduction substance.

As previously stated, the aim of this study was to try to find some method of inhibiting the action of the virus and to prevent another take after the person or animal had been successfully inoculated. The ears of white rabbits were found to be the most satisfactory site for inoculation and observation, so a series of rabbits were used with controls in each group.

Sulfanilamide and neoprontosil (disodium 4-sulfamidophenyl-2'-azo-7'-acetylamino-1'-hydroxynaphthalene-3', 6' disulfonate) were the first drugs used. The rabbits were inoculated with the vaccinia virus. At the first appearance of satisfactory inoculation, usually the second to the fourth day, the drugs were given. In all the animals the pustules followed the same course and size as in the controls. To another series of rabbits the drugs were first administered for several days until they became cyanosed, indicating a high concentration of the drugs. Then they were inoculated with vaccinia virus, and in all a satisfactory take occurred. Therefore, we concluded that the action of the virus was not inhibited by sulfanilamide or neoprontosil. Sulfapyridine was used in the same way, but it did not inhibit the action of the virus.

A number of drugs, such as tincture of iodine, metaphen and silver nitrate, were used as follows: After the epidermis was abraded, the drug was applied to the site and then the virus. No takes occurred when the tincture of iodine or metaphen was used. In another series the ears were vaccinated in the usual way, and wet dressings with ascorbic acid were applied. In these, no take occurred.

Recently Friedenwald¹⁵ reported that ascorbic acid is normally found in both the blood and the aqueous and that the concentration in the

14. Kligler, I. J., and Bernkopf, H.: *Nature*, London **139**:965-966, 1937.

15. Friedenwald, J. S.: *Tr. Am. Ophth. Soc.*, 1939, to be published.

aqueous is ten times greater than it is in the blood. Since ascorbic acid has been found to inhibit the action of the vaccinia virus, administration of ascorbic acid to increase the saturation in the blood and the aqueous might possibly offer some added protection to the cornea.

SUMMARY

No cases of vaccinia with ocular involvement have been seen since this study was started. Therefore, no definite conclusion as to the value of these measures in the prevention of corneal involvement in the human being has been made. However, they suggest a method of treatment that might offer some promise of preventing corneal involvement. It is most distressing to watch the progress in such cases, while treating the lesion symptomatically and hopefully praying that the cornea will not become involved.

In the treatment of vaccinia with ocular involvement it would seem rational to use *some of the following measures*: First, one should avoid trauma that might cause inoculation of the cornea, and great care should be exercised in opening the swollen lids. For young children, cuffs should be used to prevent them from bending the elbows and thus rubbing the eyes with the fingers. Such cuffs may also be used to prevent them from touching or scratching the original vaccination site, and thus carrying the virus to the eyes.

As soon as the lesion on the lids is diagnosed, potassium permanganate, tincture of iodine, metaphen or mercury bichloride should be applied to the lesion.

Ascorbic acid may be administered generally and locally.

The patient may be inoculated again with the vaccinia virus to increase the immunity or virulicidal substance.

Antivaccinal serum may be applied locally to inactivate the virus present on the margins of the lid and in the conjunctival secretion. The immunity may also be increased by the injection of immune serum.

EXPERIMENTAL RETINOBLASTOMA

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The present investigation was suggested by previous experimental work on the effect of carcinogenic substances on neural structures.¹ It was thought that the retina, as an easily accessible neural organ, should lend itself better to such study than the brain itself.

The first attempt to study the effect of carcinogenic substances on the eyeball was made by Schreiber and Wengler.² On the basis of the discovery of B. Fischer, who had demonstrated the carcinogenic properties of scarlet red, they injected a concentrated solution of this dye in olive oil into the anterior and posterior chambers of the eyes of rabbits and dogs. They described the new formation of large ganglion cells in the retinas of these animals about one hundred and twenty days after the injection and concluded that adult cells of the retina are able to divide mitotically. Unfortunately, no actual photomicrographs were added to their publication. It is surprising that despite the progress made in the experimental study of tumors in general no reports could be found in the literature of the last decade dealing with the application of these new discoveries on the eye.

In the present investigation two carcinogenic substances were originally used, 1,2,5,6-dibenzanthracene in lard solution and styryl 430 (2[p-aminostyryl] 6 [p-acetylaminobenzoylamino] quinoline methoacetate) in aqueous solution. The latter did not give any results. It soon became evident that in the control rats lard alone produced intense proliferation of the different tissues, corroborating the carcinogenic action of lard, olive oil and other fatty mediums in other organs as

Read as a preliminary paper at a meeting of the Chicago Ophthalmological Society, May 8, 1939.

From the Institute of Neurology and the Department of Ophthalmology, Northwestern University Medical School.

1. Weil, A.: Experimental Production of Tumors in the Brains of White Rats, *Arch. Path.* **26**:777 (Oct.) 1938.

2. Schreiber, L., and Wengler, F.: Ueber Wirkungen des Scharlachöls auf das Auge speziell auf die Netzhaut, Mitosenbildung der Ganglienzellen, *Arch. f. Ophth.* **74**:1, 1910.

reported by Burrows, Hieger and Kennaway.³ Consequently, the attempt was made to fractionate lard into different compounds by fractionate distillation in vacuo (up to 250 C.) and by saponification and to test the action of the different substances on the eye of the rat. Other substances which were tested were olive oil and iso-oleic acid (supplied by C. J. Farmer).

Altogether, until the date of submission of this paper 34 rat eyes have been studied, the rats having a maximal survival period of eleven months. The present paper is concerned mainly with experiments of injecting lard and olive oil into the corpus vitreum of the rat eye.

TECHNIC

The lard (Swift's) was heated in vacuo (water suction pump) to a temperature of 250 C., and the distillate was separated from the residue. The different fractions of the distillate did not prove to be active, and, therefore, in the final experiments the residue only was used. The material to be tested was sterilized for one hour at 120 C. and then cooled to about 40 C. White rats weighing between 150 and 180 Gm. were used. After the induction of light ether anesthesia, the injection was made through the sclera and the retina into the corpus vitreum, a long needle (no. 27) being used. The number of drops injected could easily be controlled. The rats recovered soon, and except for a mild transitory conjunctivitis no external lesions remained. The transparent fat soon changed into a yellowish granular mass, and the eye became atrophic. The rats were killed at different periods; the eyeballs were fixed in solution of formaldehyde U. S. P. or in Bouin's solution. They were embedded in paraffin, cut in a sagittal plane at 10 microns in serial sections and stained with different methods.

RESULTS

One week after the injection of superheated lard a cellular granulation tissue appeared which filled the corpus vitreum. At the points of contact with the retina the cells of the ganglion cell layer had increased considerably in number. In sections impregnated with a silver stain the majority of these newly formed cells showed a large vesicular nucleus, surrounded by argyrophilic cytoplasm (fig. 4 B). In preparations stained with cresyl violet the nucleus contained a fine, dustlike chromatin and a tiny nucleolus. While normally only one layer of cells is present with nuclei measuring from 6 to 9 microns, after injections of superheated lard there appeared several layers of large cells with nuclei measuring from 10 to 12 microns (fig. 4 A).

At the early stages after the injections of superheated lard, which were studied at eight, fourteen and twenty-nine days, respectively, the cytoarchitecture of the retina was well preserved, and there was only

3. Burrows, H.; Hieger, I., and Kennaway, E. L.: Studies in Carcinogenesis, J. Path. & Bact. 43:419, 1936.

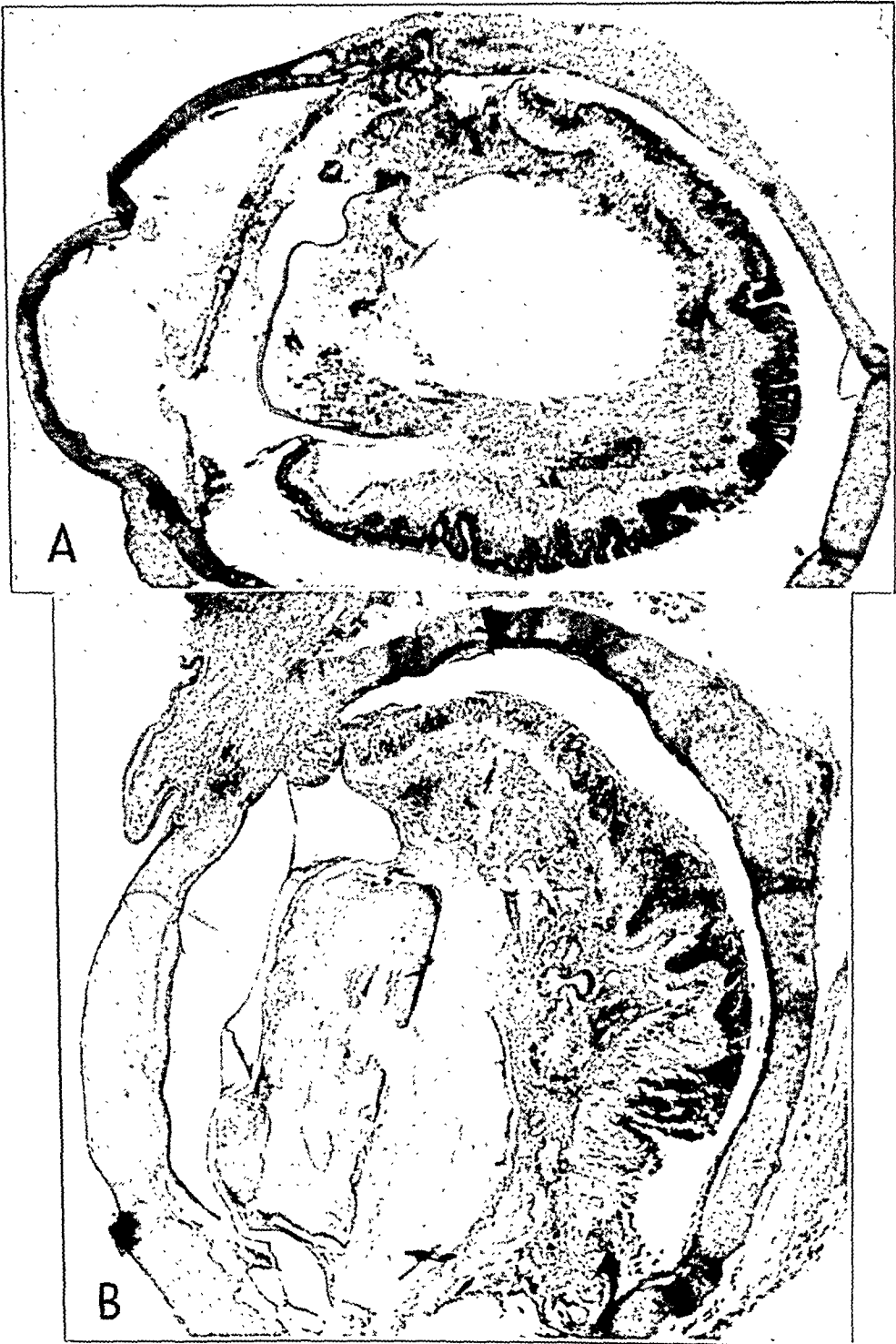


Fig. 1.—Sagittal section through a rat eye into which superheated lard had been injected. Van Gieson's stain. *A*, appearance one hundred and twenty-nine days after the injection. Magnification, $\times 24$. The retina is hypertrophic, and the corpus vitreum is replaced by granulation tissue. The lens has been removed. *B*, appearance one hundred and fifty-four days after the injection. Magnification, $\times 26$. There is marked hypertrophy of the retina with loss of differentiation between the inner and the outer nuclear layer. The granulation tissue contains large multinuclear cells surrounding acicular spaces.

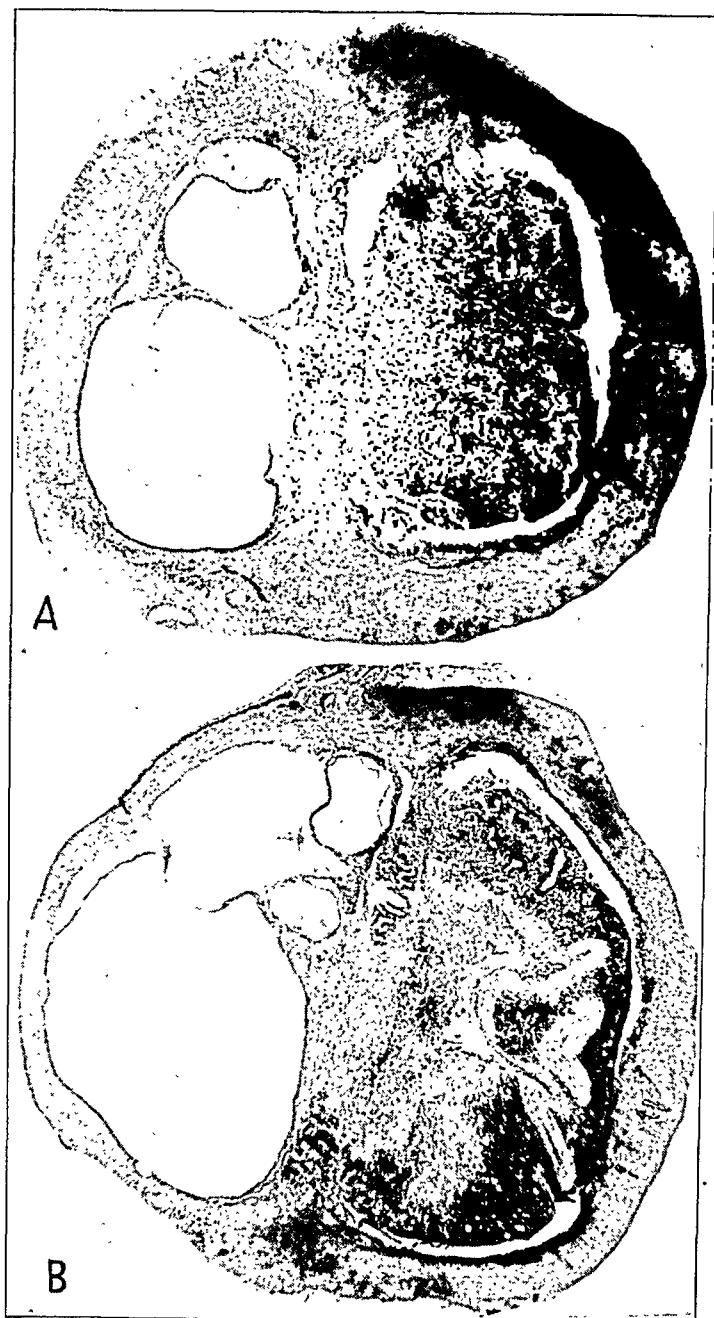


Fig. 2.—Sagittal section through a rat eye into which superheated lard had been injected two hundred and eighteen days previously. Van Gieson's stain; magnification, $\times 23$. The lens had been removed. *A*, section through the outer third of the eyeball. *B*, section through the center. There is a considerable proliferation of the retinal cells which fills out the space between the sclera and the lens. The former granulation tissue has been transformed into an atrophic connective tissue scar. The outlines between the inner and the outer nuclear layer have disappeared, and cell masses are seen invading the inner reticular layer.



Fig. 3.—Section through the retina of a rat eye into which olive oil had been injected one hundred and eighty-nine days previously. Van Gieson's stain. In *A*, the hypertrophic retina is folded, and long tubes are formed, lined at the inner surface by rods and cones. Transverse sections of these tubes give the impression of rosettes, two of which are seen in the right upper corner. They are surrounded by a cluster of small nuclei and contain large scavenger cells. Magnification, $\times 300$. In *B*, germinal centers appear in the inner nuclear layer with great numbers of mitotic figures. Newly formed cells are seen migrating from the outer into the inner nuclear layer. Magnification, $\times 375$.

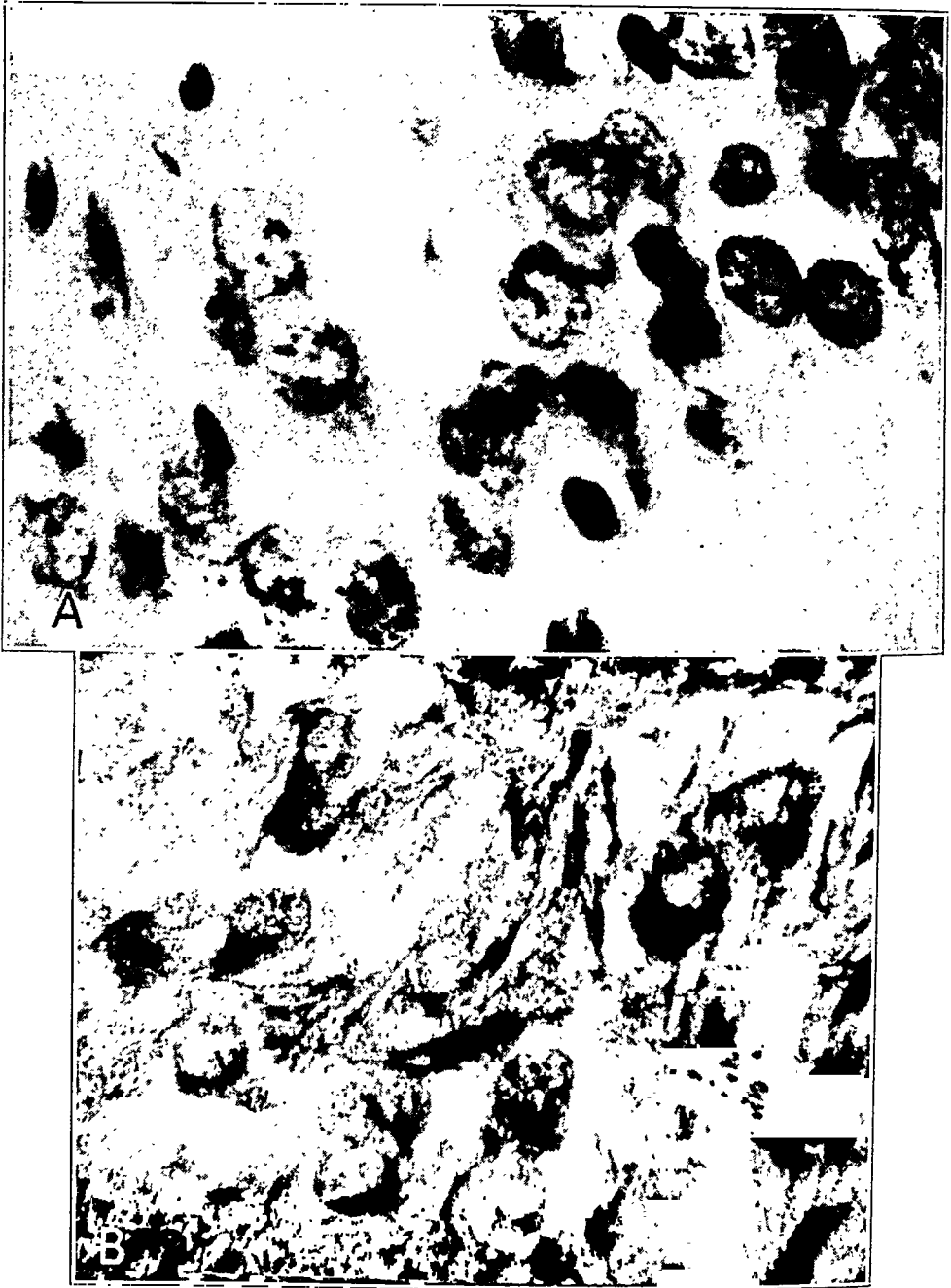


Fig. 4.—Proliferated cells in the ganglion cell layer. *A*, a rat eye into which iso-oleic acid had been injected one hundred and thirteen days previously. Cresyl violet stain; magnification, $\times 1,200$. The large nuclei contain a fine granular chromatin and a nucleolus. The cytoplasm stains well but does not show a definite structure. *B*, a rat eye into which olive oil had been injected one hundred and eighty-nine days previously. Davenport's stain; magnification, $\times 1,490$. The vesicular nuclei are surrounded by an argyrophilic cytoplasm.

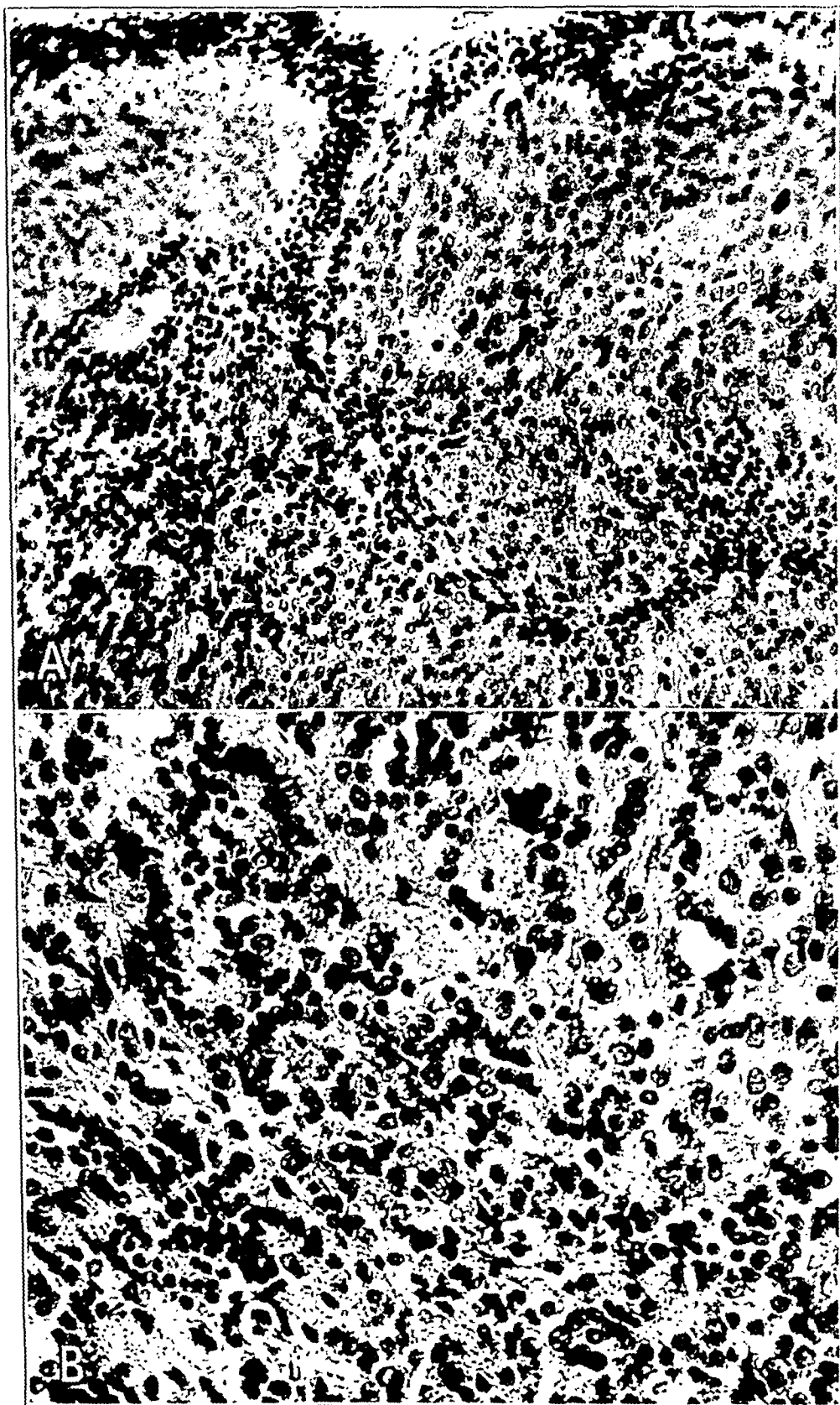


Fig. 5.—Retina of a rat eye into which superheated lard had been injected two hundred and eighteen days previously (same eye as shown in fig. 2). Van Gieson's stain; magnification of *A*, $\times 250$; of *B*, $\times 480$. The newly formed cells are seen invading both nuclear layers and the inner reticular layer. They have a tendency to cluster around blood vessels or to form circles around structureless tissue.

a mild edema at the points of contact with the granulation tissue. In the latter there appeared around the third week giant multinuclear cells, rich in cytoplasm, which surrounded acicular spaces, like those described in brains of rats into which lard was injected.¹ After three months the rods and cones and the outer and inner nuclear layers were well



Fig. 6.—Same eye as in figures 2 and 5. Cresyl violet stain; magnification, $\times 1,720$. Numerous mitotic figures are seen at different stages.

preserved and well separated. There was, however, a considerable increase in the size of the retina as a whole to two to three times its original size, of the inner reticular layer and of the number of cellular elements. At this stage ten and more rows of nuclei could be counted

in the inner nuclear layer, as compared with a normal figure of five rows. In addition, the size of the nuclei of the inner nuclear layer had increased from normally 5 or 6 microns to 8 or 9 microns (fig. 1 *A*).

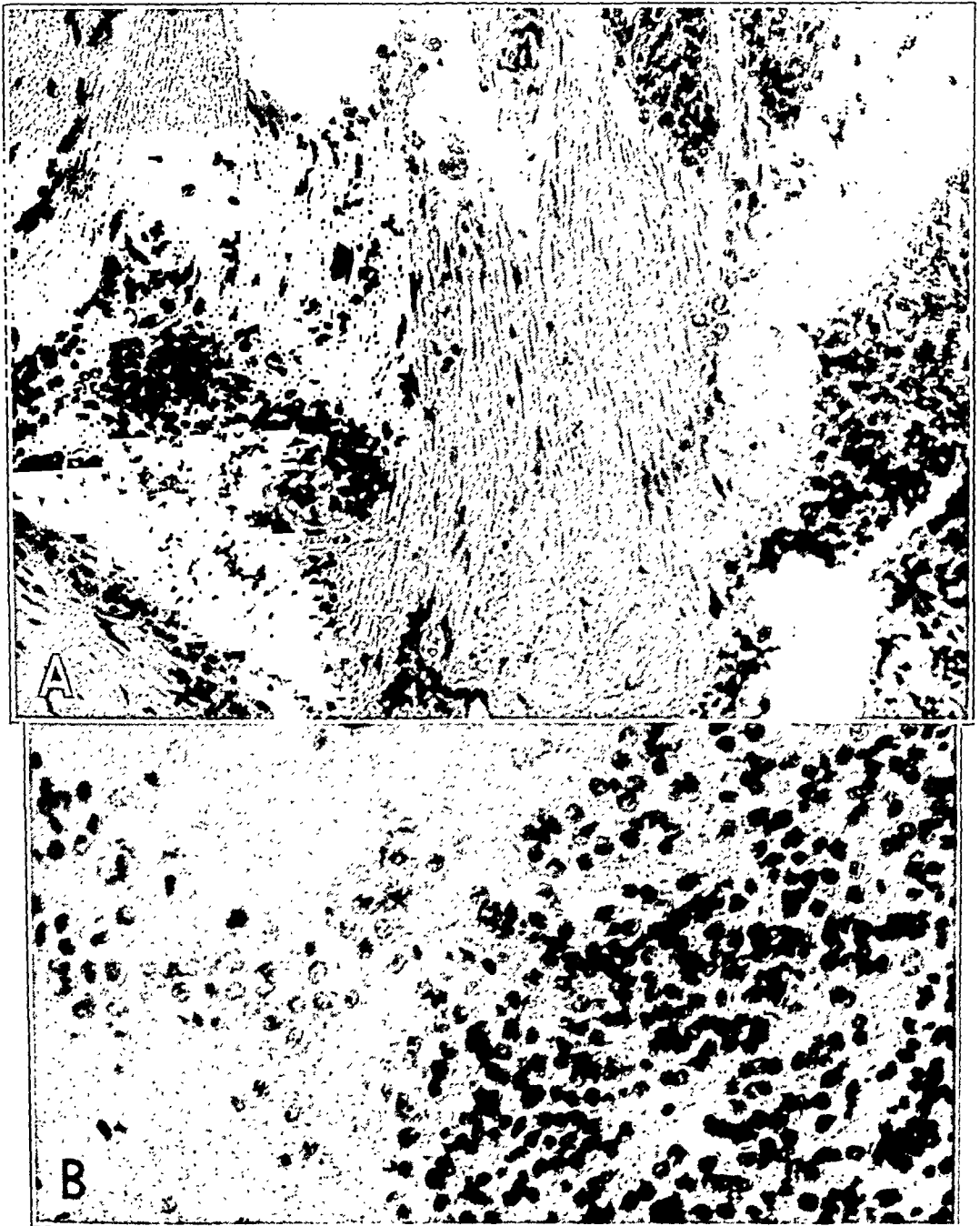


Fig. 7.—Same eye as in figures 2, 5 and 6. Van Gieson's stain. *A*, newly formed retinal cells are seen invading the optic nerve. Magnification, $\times 203$. *B*, newly formed cells are seen invading the inner reticular layer. Magnification, $\times 328$.



Fig. 8.—*A*, section through the normal retina of a rat eye. Van Gieson's stain; magnification, $\times 485$. *B*, section through a human retinoblastoma. Hematoxylin-eosin stain; magnification, $\times 44$. Part of the retina has retained its normal appearance. The tumor seems to originate from the nuclear layer.

Frequently rosettes were found surrounded by the tiny cells of the outer nuclear layer. Their formation is easily explained if one studies figure 3. The increase in the size of the retina necessarily leads to a process of folding and tubule-like formations, lined inside by a layer of rods and cones. The latter gradually disintegrated, and their debris was taken up by large scavenger cells, which may be seen in the center of the two rosettes at the right of the illustration. The smaller cells surrounding the rosettes proliferated, and around the fifth month after the injection numerous mitotic figures appeared (fig. 6). The newly formed cells were seen migrating into the inner nuclear layer, and the

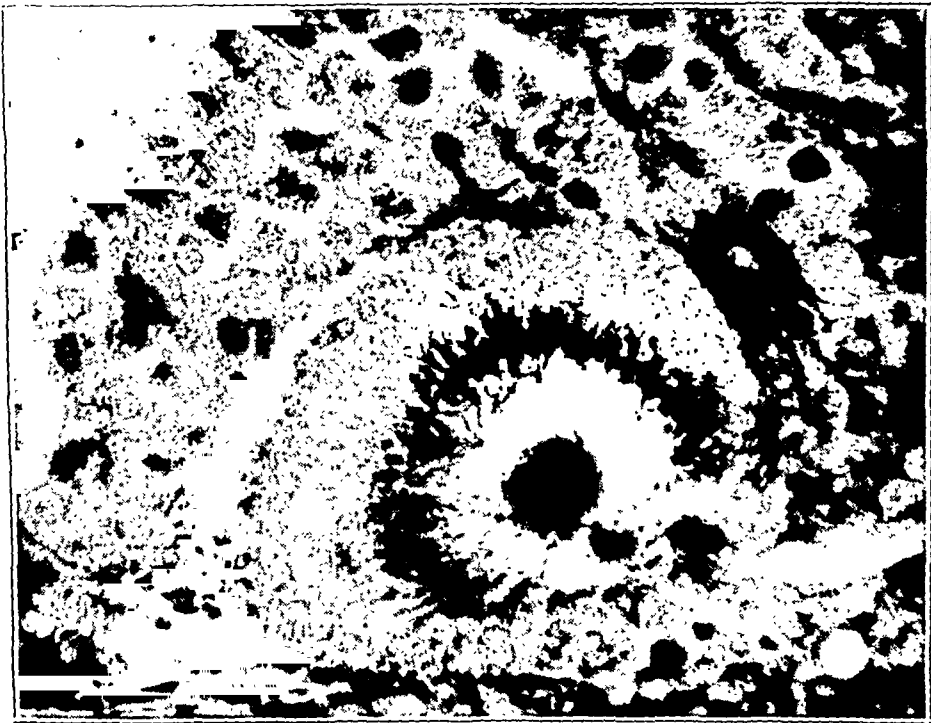


Fig. 9.—Same eye as in figures 3 and 4 *B*. Davenport's stain; magnification, $\times 630$. A typical rosette is seen with a large phagocytic cell in the center, surrounded by rods and cones.

separation between the two layers gradually disappeared. About the sixth month after the injection there began a migration of cells into the inner reticular layer (fig. 7 *B*), and finally the whole retina was transformed into a cellular structure, which expanded into the posterior chamber after the shrinkage of the original granulation tissue and the connective tissue scar which replaced it (fig. 2). In this tumor formation there was in places a tendency of cells to cluster around blood vessels or to form circles around a structureless area (fig. 5 *B*).

The papilla of the optic nerve showed swelling in the early stages. During the seventh month proliferated retinal cells were seen invading the optic nerve (fig. 7 *A*). The ciliary body showed marked hypertrophy

at about the fourth month after the injection; both papillae as a whole as well as the covering epithelium were enlarged. In 1 case a remarkable proliferation of the epithelium was observed after injection of superheated lard with the formation of detached long rows of epithelial cells. The iris showed early cellular infiltration, which persisted during the later stages. The lens in some cases underwent early degeneration. Its capsule appeared thickened, and the interior was filled with a dense accumulation of phagocytic cells. In other cases it merely was shrunken, and its structure was well preserved (fig. 1). The choroidea of the early stages was shrunken, though the pigmented cells were preserved.



Fig. 10.—Same eye as in figure 1 *B*. Van Gieson's stain; magnification, $\times 230$. Hyperplasia of the ciliary body is evident.

Five months after the injection its blood vessels became wider, but there was apparently no proliferation of the cells of the pigmented cell layer. The sclera and cornea were not changed to any marked degree, except for a mild thickening observed during the later stages.

The results following injections of olive oil and iso-oleic acid were not as uniform as those following injections of superheated lard. Iso-oleic acid produced an early inflammatory reaction, which in 1 case destroyed the retina. In another case in which olive oil was injected, no proliferative changes were found seven months after the injection, though the possibility had to be considered that most of the oil escaped

after the injection. In 1 case active proliferation and mitosis of retinal cells was observed one hundred and eighty-nine days after the injection of olive oil.

COMMENT

If one compares the present experiments with the early attempts of Schreiber and Wengler to produce tumors by injecting scarlet red in olive oil, their failure may be explained in two ways. First, they made the injections into the anterior chamber in most of their experiments and there was no direct contact between the carcinogenic substance and the retina. Second, they did not extend their experiments long enough but interrupted them at one hundred and twenty days. Judging from the present observations, there is no doubt that they actually observed the beginning of mitotic divisions; unfortunately, no actual photomicrographs were added to their paper. But the preceding descriptions demonstrate that active mitosis and neoplastic transformation begin to appear after the fifth or the sixth month only. This experience is in contrast to that obtained in experiments in which cutaneous tumors followed injections of dibenzanthracene, about four months apparently being sufficient to produce an active growth. Brain tissue seems to be still slower in responding to carcinogenic agents.

It is difficult to state which of the two nuclear layers of the retina showed the more active cellular proliferation. It seemed that the inner layer first increased in width, but that the outer layer participated with active mitosis only during about the sixth month. The experimental production of the "Wintersteiner rosettes" is not completely in agreement with the commonly held opinion that in the human eye they are formed during the neoplastic process and represent primitive neural tubes or a primitive optic vesicle. It was previously pointed out that they are formed early after the injection of superheated lard as invaginations of the enlarged retina. They imitate tubular formations, lined by rods and cones. The latter may persist within such a rosette for some time before they disintegrate. The large scavenger cells within the rosettes have been described as giant astrocytes or ganglion cells which send out fine processes. Sections impregnated with silver stain, however, demonstrate that these processes are fine, cilia-like extensions arising from the inner wall of the rosette. Furthermore, such preparations demonstrate clearly that two thin membranes separate the rods and cones from the smaller nuclei and the latter from the outer nuclear layer, respectively (fig. 9).

Attempts to impregnate the neoplastic cells with silver stains were not successful. Occasionally a tiny layer of argyrophilic cytoplasm could be seen around the vesicular nuclei or a short process of the cytoplasm. Impregnation of the proliferated cells of the ganglion cell

layer was more successful and demonstrated clearly newly formed neurons (fig. 4). No marked gliosis could be seen in preparations stained by the Holzer method, even in experiments of seven months' duration. There was also no increase in vascularization, and no necrotic areas had been formed up to this stage.

While in regard to the early experiments of up to five months' duration some doubt might exist whether one should classify the retinal proliferation as "granulomatous" or "traumatic reaction," there is no doubt that superheated lard induces a true neoplastic process six and more months after its injection into the corpus vitreum of the rat eye. The term retinoblastoma instead of neuroblastoma of the retina has been preferred for this tumor formation, in adherence to Verhoeff's terminology, in order to emphasize the primitive character of the proliferated cells.

SUMMARY

Injections of superheated lard and of olive oil into the corpus vitreum of the eyes of white rats led to neoplastic disease of the retina. The cells of the ganglion cell layer which were in direct contact with the injected substance showed active proliferation during the first month.

Both the inner and the outer nuclear layer responded with cellular proliferation, and active mitosis appeared in the latter about the sixth month after the injection. At the same time invasion of the optic nerve by proliferated retinal cells was observed.

The term retinoblastoma has been applied to this tumor formation in order to describe the primitive character of the proliferated cells.

GLIOMA OF THE OPTIC NERVE

A CRITICAL REVIEW; REPORT OF TWO CASES, WITH AUTOPSY
OBSERVATIONS IN ONE

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AND

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DURHAM, N. C.

While 1 or 2 cases of primary glioma of the optic nerve are reported each year in the literature, it is the clinical aspect that almost always predominates in these reports. Relatively little has been said regarding the pathologic study and classification of these tumors¹ since the introduction of the impregnation methods of Cajal and del Rio-Hortega. Save for Dandy's paper² in 1922, there has been no critical evaluation of the surgical procedures. It is, then, with the pathologic aspect and also with the operative approach that this paper is chiefly concerned.

In general, these tumors are divided into two large groups: (1) the dural endotheliomas or meningiomas, which arise from the fibrous sheath of the nerve, and (2) the gliomas, which arise from the neuroglial elements within the nerve itself.

Clinically, the meningiomas seem to be more malignant than the gliomas, a fact recognized by Hudson³ in 1912. Twenty-nine cases were cited in his paper. Nineteen of the patients were followed for less than one year. Of the remaining 10, 2 died of meningitis at operation, 4 died of recurrences and 4 were followed for from one to six years without symptoms of recurrence.

Two of the cases in which there were recurrences are cited. In the case reported by Byers,⁴ recurrence caused death nine years after

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1. del Rio-Hortega, P.: Estructura y sistematización de los gliomas y paragliomas, *Arch. españ. de oncología* 2:569, 1932; *Anatomía microscópica de los tumores del sistema nervioso central y periférico*, Madrid, S. A. Blass, 1934, p. 154.

2. Dandy, W. E.: Prechiasmal Intracranial Tumors of Optic Nerves, *Am. J. Ophth.* 5:169, 1922.

3. Hudson, A. C.: Primary Tumours of the Optic Nerve, *Roy. London Ophth. Hosp. Rep.* (pt. 3) 18:317, 1912.

4. Byers, W. G. M.: Primary Intradural Tumors of the Optic Nerve: Fibromatosis Nervii Optici, in *Studies from the Royal Victoria Hospital*, Montreal, Toronto, J. A. Carveth & Co., 1901, no. 1, p. 3.

primary removal of the tumor; there was a recurrence in the orbit one year after the first operation, at which time exenteration was done. The patient, a girl, lived eight more years, and autopsy revealed extensive intracranial involvement.

The other case is reported by Pagenstecker.⁵ A local recurrence appeared twenty-five years after the original operation, death from thrombosis of the cavernous sinus following the second removal. At autopsy it was found that the tumor had not advanced as far as the chiasma.

Of considerable interest, though not of importance to this paper, are the bilateral intracranial meningiomas involving the optic nerve described by Schott⁶ and Dandy.²

Among the cases of glioma, we found nothing comparable to these cases of meningioma in which recurrence took place nine and twenty-five years later. The course seems rather to be benign. In Lundberg's case 1⁷ an oligodendrocytoma was thought to have been incompletely removed, and the patient was in good health seventeen years later. The patient in Byers' case 1⁴ was alive and well after fifteen years, although at operation the growth was found to extend up to the optic foramen. The patient in Seefelder's case 1⁸ showed no evidence of recurrence in ten and one-half years, nor did de Schweinitz' patient⁹ in seven years. It is not known whether in the last 2 cases the growth was thought to be completely removed. Only in the case of Barraquer¹⁰ was there intracranial extension after an appreciable lapse of time (nine years).

Such, then, was the general concept of the problem under which the following 2 cases were handled.

REPORT OF CASES

CASE 1.—*History*.—M. H., an 8 year old Negro girl, was admitted to the hospital on June 23, 1936, because of exophthalmos of the right eye of two years' duration. The past and family histories were unimportant. The exophthalmos

5. Pagenstecker, A. H.: Ueber Opticustumoren, *Arch. f. Ophth.* **54**:300, 1902.

6. Schott: On Some Affections of the Optic Nerve, *Arch. Ophth.* **6**:276, 1877.

7. Lundberg, A.: Le gliome primitif du nerf optique et du chiasma des nerfs optiques, *Arch. d'ophth.* **1**:97, 1937.

8. Seefelder, R.: Beiträge zu den Gliomen des Sehnerven, *Wien. klin. Wchnschr.* **44**:838, 1931.

9. de Schweinitz, G. E.: A Contribution to the Subject of Tumors of the Eyelid and Orbit, *Tr. Am. Ophth. Soc.* **14**:341, 1915-1916.

10. Barraquer, J.: Mixoma quistico del nervio optico de la papila y retina derechas y de la cavidad craneal y órbita izquierda, *Arch. de oftal. hispano-am.* **2**: 132, 1902.

was gradual in onset and progressive. There had been no ocular pain or headache. Vision was noted to have been lost about two months before the patient came to the hospital. There had been no difficulty with the left eye. The patient's general health had been good.

Examination.—There was marked exophthalmos of the right eye. Vision was limited to perception of light. Motion was partially limited on internal rotation. The globe could be entirely displaced, but no distinct mass could be felt in the orbit. However, the retrobulbar space appeared to be filled with nonpulsating undifferentiated tissue. The lids, conjunctivas, sclera, cornea, iris and media were normal. The pupil was partially dilated but reacted consensually. The right optic disk showed primary atrophy.

Vision in the left eye was 20/20, and examination otherwise gave entirely negative results.

General physical and neurologic examinations gave negative results. The Wassermann reaction was negative, and the blood and urine were normal. Roentgenograms of the skull showed the right optic foramen to be dilated, measuring 10 mm., while the left measured but 4 mm. There was no evidence of bone erosion.

A diagnosis of retrobulbar tumor was made, and the patient was prepared for operation.

Operations.—Exploration of the orbit was done with the patient under ether anesthesia. A small hard tumor measuring 5 by 2 cm. was removed; it was entirely within the muscle cone and extended to the apex of the orbit, apparently encapsulating the optic nerve. Because of the type of tumor first reported, craniotomy for removal of the intracranial portion of the tumor was deemed necessary, and this was attempted by a member of the neurosurgical service three weeks after the first operation. A right anterior trephine opening was made, but when the dura was incised excessive bleeding was encountered. Further operative procedures were postponed until one week later, when a firm, circumscribed, pinkish tumor, 1.5 by 1 cm. in size, was found to protrude from the optic foramen posteriorly toward the optic chiasma. It was not attached except at the foramen. The optic nerve was not involved as far as the chiasma but seemed to fray out into the tumor and was easily divided with the foramen hook. The tumor was readily removed with a sharp curet. It was felt that some of the tumor remained in the optic foramen, and because of inadequate exposure attempts were made to remove it with a curet. Considerable arterial bleeding resulted from this procedure, and it was necessary to insert a pack to control it. After the bleeding was controlled, another attempt was made to remove the tumor from the optic foramen, but bleeding again occurred, and the operation was terminated after the hemorrhage was controlled with packs. The wound was closed, and the patient returned to the ward in poor condition. She failed to respond after the operation and died six hours later. Autopsy was done, and the results are reported as follows:

Pathologic Study.—The first surgical specimen was a soft, conical, encapsulated tumor mass, measuring 5 cm. in length and varying from 0.6 to 2 cm. in diameter. The cut surface was uniformly grayish white. In the region of the apex of the conical mass the optic nerve was seen, surrounded by a collar of neoplastic tissue (fig. 1A). As further sections were made, the nerve gradually became lost in the mass at the base of the tumor.

The second surgical specimen consisted of several fragments of soft grayish white tissue, the largest of which measured about 1 cm. in diameter. Both specimens were fixed in a 10 per cent dilution of solution of formaldehyde U. S. P.

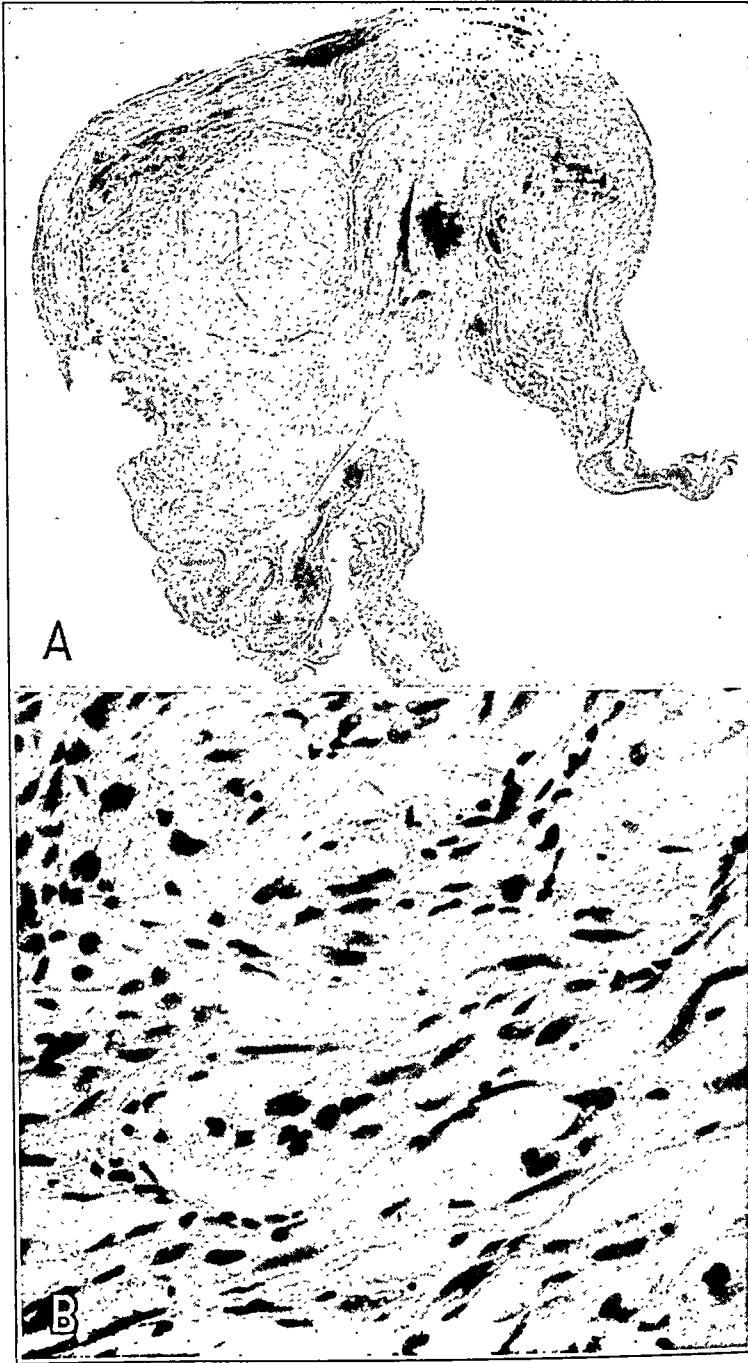


Fig. 1 (case 1).—*A*, low power photomicrograph showing the orbital portion of the optic nerve surrounded by tumor which fills the intervaginal space. The torn dura is an artefact. *B*, high power photomicrograph of the same section.

and stained with hematoxylin and eosin and with Mallory's connective tissue stain. A preliminary diagnosis of "benign fibrous tissue tumor" was made in each instance.

Autopsy observations of significance were limited to the cranial cavity. There were extensive hemorrhage and maceration of the base of the brain anteriorly. A small fragment of a yellowish gray tumor tissue was found in the region of the optic foramen. This was fixed in Helly's fluid and in a solution of formaldehyde and ammonium bromide.

Microscopically, all the specimens presented much the same picture (fig. 1 *B*). Save at the periphery, where a few degenerated nerve fibers were seen, the optic nerve was not remarkable. Surrounding this was a collar of dense tissue, which at first suggested a fibrous tissue tumor. Dense irregular bands of connective tissue divided the tumor into irregular areas, the picture resembling the coarsely reticulated type of Verhoeff.¹¹ The tumor cells were elongated, presenting small oval nuclei. Fine and coarse glia fibrils coursed irregularly through the mass. Many psammoma bodies were present. Sections impregnated with silver and gold stains were not entirely satisfactory but suggested a spongioblastoma polare.

The point of origin of the tumor in the nerve was not demonstrated, nor were the relations of the nerve to the intracranial portion of the tumor made entirely clear.

The case is registered at the Army Medical Museum (accession no. 50 514).

CASE 2.—History.—M. C. (fig. 2 *A*), a 3 year old Negro girl, was seen in the pediatric clinic on Jan. 11, 1939, because of unilateral exophthalmos of the right eye of eight months' duration. The family and past histories were entirely non-contributory. Two years before the child's entry the parents had noted an external strabismus of the right eye and sixteen months later a forward and downward displacement. The child had repeatedly complained of pain in the right eye for eight months, and vision seemed to be diminished. The day before the child's entry to the hospital bilateral swelling of the parotid glands developed, which prohibited admission to the hospital until February 2.

Examination.—Complete physical and neurologic examinations of the patient gave negative results except for the findings in the right eye. The left eye was normal. Visual acuity could not be tested. The right eye was displaced downward and outward; motility was greatly limited. The conjunctivas appeared normal. In the superior nasal quadrant there was a faint bulge in the sclera. Palpation revealed no mass or erosion of the orbital rim. The pupil reacted to light. The intraocular tension was normal.

On ophthalmoscopic examination the media were found to be clear. The disk could not be seen because of a large, grayish, elevated oval mass (fig. 2 *B*) which extended temporally to the macular region and about the same distance in other meridians. The mass appeared to be cystic and undulatory. There were no retinal vessels overlying it. These appeared to dip beneath the margin of the mass as they approached the disk. The arteries appeared larger than usual, the inferior nasal artery in particular being about twice the normal diameter. This artery was not accompanied by a vein, and about 4 disk diameters from the disk it suddenly dipped into the sclera. A short distance peripherally there was a small

11. Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of Optic Nerve: A Histologic Study of Eleven Cases, Including Case Showing Cystic Involvement of Optic Disc, with Demonstration of Origin of Cytoid Bodies of Retina and Cavernous Atrophy of Optic Nerve, *Tr. Sect. Ophth., A. M. A.*, 1921, p. 146.

area of old chorioretinitis. The remainder of the retina was normal except for the richness of the arteriolar coloring and the increased caliber of the walls of the vessels.

Roentgenograms of the optic foramens showed the right to be 9 mm. in diameter and the left 5 mm. Films of the skull and sinuses were normal.

Operation.—The day after the child's admission the globe and a large chocolate-colored, spindle-shaped tumor were removed, ether anesthesia being used. The

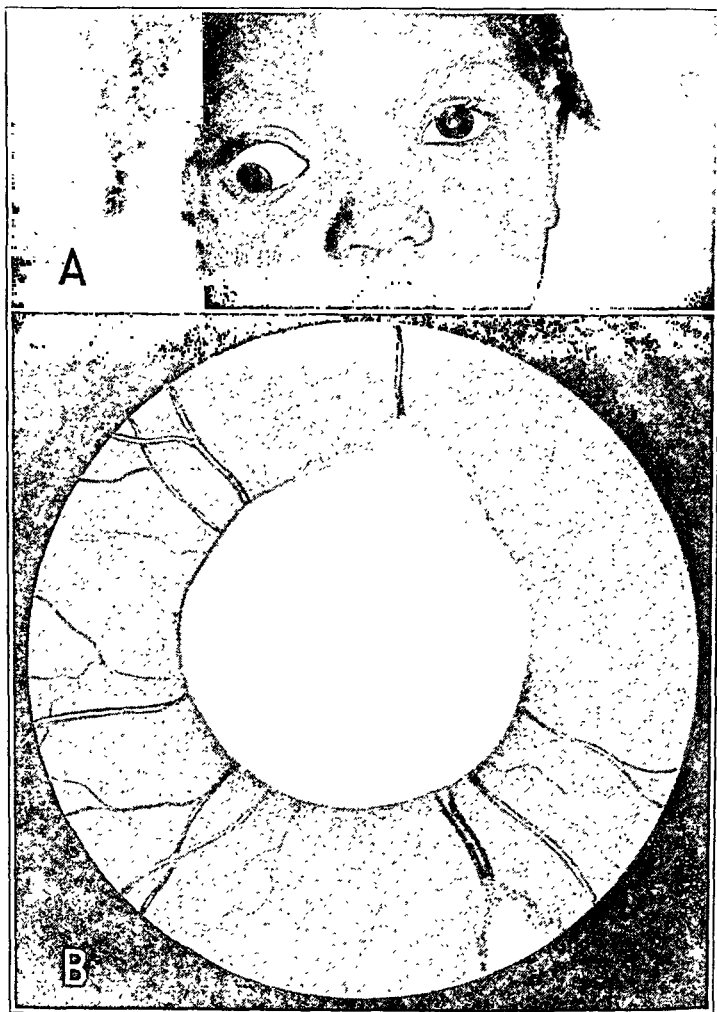


Fig. 2 (case 2).—*A*, photograph of the patient, showing the exophthalmos downward and laterally. *B*, photograph of a drawing of the tumor of the disk. Compare this with figure 3 *B*.

orbital portion of the tumor could not be felt until the globe was removed. This portion of the tumor measured 4 cm. in length and 1 cm. in breadth. The smaller end of the tumor extended into the apex of the orbit. No unusual bleeding was encountered.

The postoperative course was entirely uneventful, and the patient was discharged from the hospital on the twelfth day.

The patient returned for a check-up two months later. Roentgenograms of the skull were normal, and ophthalmoscopic examination gave negative results.

Pathologic Study.—The eyeball measured 2.5 cm. in the transverse diameter and grossly presented nothing remarkable. The optic nerve was cut level with the sclera and was not enlarged at this point. The eye was fixed in Bouin's fluid and sectioned after hardening.

The tumor was a sausage-shaped mass (fig. 3 *A*), measuring 3.6 cm. in length and 1.4 cm. in greatest diameter. At the larger end was a circular break of 1.5 mm. in the capsule, where it had been cut from its attachment to the eyeball.

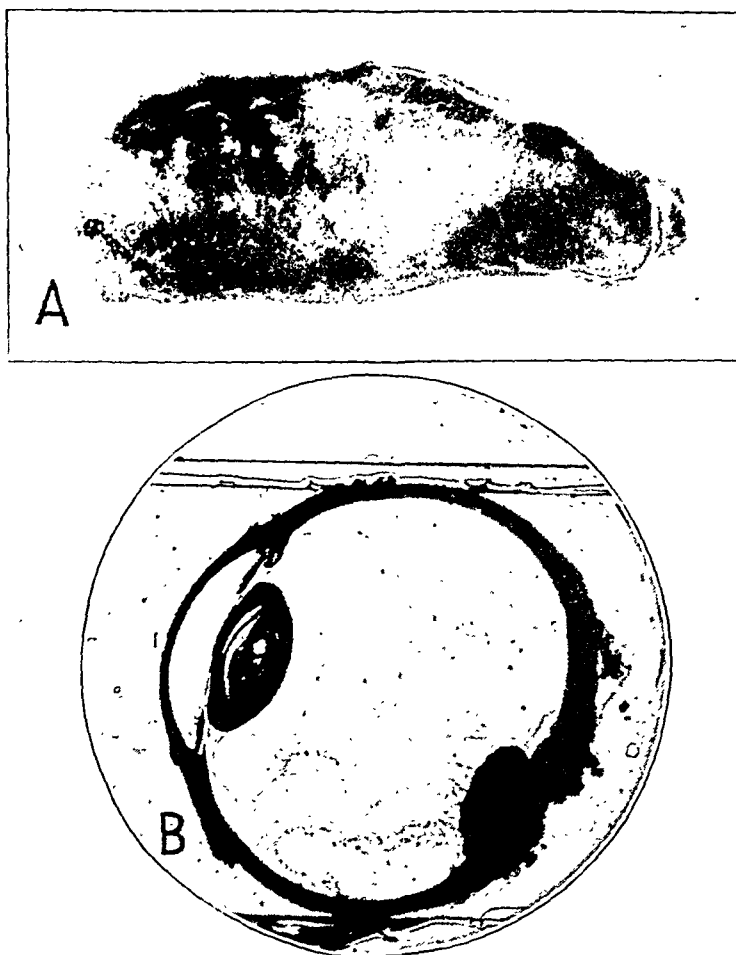


Fig. 3 (case 2).—*A*, gross appearance of the intraorbital tumor. The small teatlike process at the left is the point of attachment to the bulb. *B*, section through the globe; $\times 2$. The photograph was overexposed to bring out the areas of liquefaction in the vitreous and to demonstrate the fibrinous exudate on the surface of the tumor. Compare this picture with figure 2 *B*.

At the opposite end the thin capsule had been cut through, and the myxomatous tumor tended to extrude. Grossly, it appeared to have been incompletely removed. The cut surface presented a soft, translucent, whitish appearance in which a few longitudinal striations were noted. The tissue was fixed in solution of formaldehyde, Zenker's fluid and in a solution of formaldehyde and ammonium bromide. Sections were stained with hematoxylin and eosin, Mallory's connective tissue and phosphotungstic acid stains and Bielschowsky's silver stain and were impregnated with silver carbonate and gold chloride sublimate.

The mass of the tumor was composed of a loose reticulum of glial tissue, separated into irregular bundles by thick bands of tissue, in part fibrous and in part compressed glial material (fig. 4 *B*). The loose portion consisted largely of glia fibers, but many cells were noted, the oval nuclei of which contained a faint

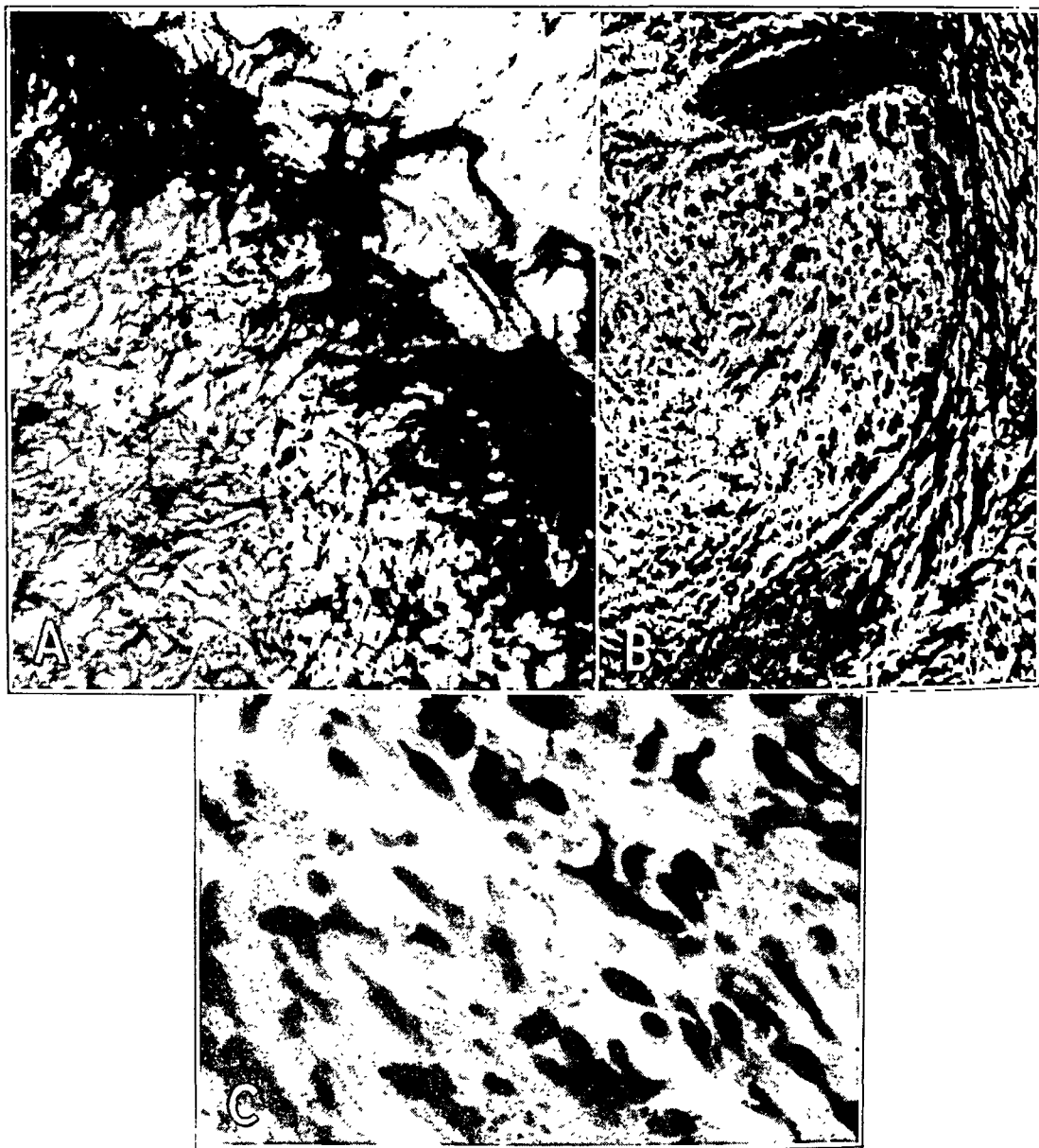


Fig. 4 (case 2).—*A*, section through the edge of the intraocular portion of the tumor. Mallory's connective tissue stain, $\times 140$. Note the looseness of the tumor, the astrocytes and spongioblast and the fibrinous exudate at the periphery of the tumor. *B*, intraorbital tumor. Hematoxylin and eosin stain, $\times 140$. Note the fibrous septum dividing the looser tumor into irregular spaces filled with glial cells and fibrils. *C*, intraorbital tumor. Fixation with solution of formaldehyde and ammonium bromide and silver carbonate impregnation, $\times 650$. Note the elongated spongioblasts.

chromatin network and several (frequently two) sharply defined chromatin dots. The cells possessed little cytoplasm, which occasionally could be seen prolonged into the glial processes. Unfortunately, no myelin stains were made, but Bielschowsky's silver stain did not reveal any axis-cylinders, nor did the hematoxylin and eosin stain suggest the presence of any nerve fibers. The gold and silver impregnations (fig. 4 C) showed the majority of the cells to be polar spongioblasts, though a few plump astrocytes were present.

Microscopic study of the eye revealed little of importance save the tumor. The vitreous (fig. 3 B) presented several cystlike areas of liquefaction. Rising from the surface of the tumor mass, numerous fibrinous strands extended anteriorly, but no actual invasion of the vitreous by tumor was noted. The hyaloid membrane was intact save over the summit of the tumor, where its outline was lost in the fibrinous material.

The disk was swollen and elevated, being 1.5 mm. in thickness. It was surmounted by a flattened mushroom-like mass of the same thickness, lying so close that at first glance it seemed to be part of the disk. However, microscopically there was seen a dividing line, formed by a reflection of the internal limiting membrane.

When the tumor was approached from the normal retina at the periphery, the rod and cone layer and the external nuclear layer were first involved, being thinned and compressed by the tumor growth. The outer reticular layer was fairly well preserved. The inner nuclear layer gradually became disorganized, showing microscopic cystlike spaces. The inner reticular and ganglion cell layers were completely disorganized. The internal limiting membrane was thrown into a series of rounded serrations, which flattened out as the slope of the tumor was ascended. In the deeper portion of the tumor, the disk, there was a large amount of fibrous tissue intermingled with the glia fibers. The upper mushroom-like portion consisted almost entirely of glia, most of the cells appearing to be bipolar spongioblasts (fig. 4 A). Many astrocytes were likewise noted. No cysts were present as in the other cases reported.

COMMENT

Since the optic nerve is not a nerve in the true sense of the word but rather an extension of the brain substance, even to its fibrous sheath, and since the studies of Marchesani¹² have shown that the same neuroglial elements are present as are found in the brain, it is only logical to suppose that the glial and fibrous tissue tumors of the nerve should be comparable to those of the brain. There are now available the histologic technics of Cajal and del Rio-Hortega, which were not available when Verhoeff¹¹ offered his microanatomic classification in 1921 (finely reticulated, coarsely reticulated and coarsely fibrillated), and the trend toward a specific qualitative analysis of these tumors is evident in the recent literature. Mehney¹³ reported 3 cases of spongioblastoma

12. Marchesani, O.: Die Morphologie der Glia im Nervus opticus und in der Retina, dargestellt nach den neuesten Untersuchungsmethoden und Untersuchungsergebnissen, Arch. f. Ophth. **117**:575, 1926.

13. Mehney, G. H.: Primary Tumor of Optic Nerve: Report of Case, Arch. Ophth. **16**:95 (July) 1936.

polare; Kiehle,¹⁴ Stern¹⁵ and Rand, Irvine and Reeves¹⁶ each reported 1 case. DeLong¹⁷ implied that the growth in his case was of this type. Goldstein and Wexler¹⁸ reported a case of spongioneuroblastoma and Santori¹⁹ a case of astrocytoma. Eickhoff,²⁰ Foerster and Gagel²¹ and Seefelder⁸ reported cases of spongioblastoma multiforme. Lundberg⁷ reported 9 cases of oligodendrocytoma, and Schreck²² and Cárdenas²³ each reported a case.

Since the term glioma no longer suffices, the differentiation into specific types of glioma must be attempted. It is felt that while many of the tumors can be accurately diagnosed by the usual glial stains, the best, and in many cases the only accurate, techniques are the impregnation methods of Cajal and del Rio-Hortega. These require special fixation in a solution of formaldehyde and ammonium bromide and experienced technical preparation, their final interpretation being based on the predominating cell type. Also, it may be noted that in our experience Mallory's phosphotungstic acid-hematoxylin stain for glia is reliable only after fixation in Zenker's fluid. Thus, of the 23 cases cited, only in that of Cárdenas²³ are the impregnation methods known to have been used, while in several others only tissue that had been fixed in solution of formaldehyde was available. We emphasize, therefore, that while there is need for a specific differentiation of these tumors so that a rational prognosis can be made (since the various types present

14. Kiehle, F. A.: Tumor of Optic Nerve: Report of Case, *Arch. Ophth.* **15**:686 (April) 1936.

15. Stern, R. O.: Tumor of Optic Nerve, Chiasma and Thalamus, *Proc. Roy. Soc. Med.* **30**:1096, 1937.

16. Rand, C. W.; Irvine, R., and Reeves, D. L.: Primary Glioma of the Optic Nerve: Report of a Case, *Arch. Ophth.* **21**:799 (May) 1939.

17. DeLong, P.: Primary Tumors of the Optic Nerve: Report of Case, *Am. J. Ophth.* **17**:797, 1934.

18. Goldstein, I., and Wexler, D.: Spongioneuroblastoma of Optic Nerve in Neurofibromatosis (Recklinghausen), *Arch. Ophth.* **7**:259 (Feb.) 1932.

19. Santori, G.: Glioma primitivo astrocitario diffuso del nervo ottico, *Boll. d'ocul.* **9**:464, 1930.

20. Eickhoff, W.: Intraneurales Wachstum eines Glioms (Nerv. opticus), *Virchows Arch. f. path. Anat.* **302**:222, 1938.

21. Foerster, O., and Gagel, O.: Ein Fall von sog. Gliom des Nervus opticus—Spongioblastoma multiforme ganglioides, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **136**:335, 1931.

22. Schreck, E.: Ein Oligodendrogliom als Beitrag zu den primären intraduralen Optikustumoren, *Klin. Monatsbl. f. Augenh.* **100**:560, 1938.

23. Cárdenas Pupo, M. D.: Contribución al estudio del oligodendrocitoma del nervio visual (Río-Hortega), *Bol. Liga contra el cáncer* **9**:196, 1934.

various degrees of malignancy), the methods by which this differentiation is reached must be carefully evaluated.

Invasion of the disk by a tumor growth of the optic nerve is rare, only 15 cases being reported so far in the literature. And although the statement has been made that meningiomas more frequently involve the disk than gliomas, only 6 cases of the former are found but, with the 1 case here reported, 10 cases of the latter are recorded.

Invasion by meningioma has been reported by Jacobson,²⁴ Goldzieher,²⁵ Baeumler,²⁶ Neame,²⁷ Schwarz²⁸ and Coston.²⁹ Hirschberg's case,³⁰ which is occasionally cited, was probably one of sarcoma of the choroid, although von Recklinghausen thought that the growth arose from the disk.

Of the gliomas, von Graefe's case³¹ in 1864 is probably the first recorded. Barraquer's case¹⁰ is especially interesting in that the patient was followed for nine years, and clinically the growth showed intracranial extension. The case of Sulzer and Rochon-Duvigneaud³² was probably one of glioma, while that of Pereyra³³ in 1914 undoubtedly was. Verhoeff,¹¹ Martin and Cushing,³⁴ Sattler³⁵ and Weigelin³⁶ reported cases in 1921, 1923, 1926 and 1931, respectively, the growth in Sattler's case being extraordinarily like that described here (case 2). Finally, using more specific terminology, Foerster and Gagel²¹ in 1931

24. Jacobson, P. J.: *Klinische Mittheilungen*, Arch. f. Ophth. (pt. 2) **10**:55, 1864.

25. Goldzieher, W.: *Die Geschwülste des Sehnerven*, Arch. f. Ophth. (pt. 3) **19**:139, 1873.

26. Baeumler, E.: *Ein Fall von Orbital- und Uvealsarkom*, Klin. Monatsbl. f. Augenh. **24**:5, 1886.

27. Neame, H.: *Tumour of Optic Nerve*, Brit. J. Ophth. **7**:209, 1923.

28. Schwarz, K.: *Zur Klinik und Anatomie der Geschwülste des Sehnerven*, Arch. f. Ophth. **135**:247, 1936.

29. Coston, T. O.: *Primary Tumor of Optic Nerve, with Report of Case*, Arch. Ophth. **15**:896 (April) 1936.

30. Hirschberg, J.: *Casuistische Mittheilungen über Geschwülste der Orbita und des Bulbus*, Klin. Monatsbl. f. Augenh. **6**:153, 1868.

31. von Graefe, A.: *Geschwülste des Sehnerven*, Arch. f. Ophth. (pt. 1) **10**:193, 1864.

32. Sulzer and Rochon-Duvigneaud: *Néoplasme du nerf optique et de la papille*, Ann. d'ocul. **149**:161, 1913.

33. Pereyra, G.: *Mixoma del nervo ottico*, Ann. di ottal. **43**:456, 1914.

34. Martin, P., and Cushing, H.: *Primary Gliomas of Chiasma and Optic Nerves in Their Intracranial Portion*, Arch. Ophth. **52**:209 (May) 1923.

35. Sattler, H.: *Die bösartigen Geschwülste des Auges*, Leipzig, S. Hirzel, 1926, p. 238.

36. Weigelin: *Ueber Gliomatose des Sehnerven*, Klin. Monatsbl. f. Augenh. **87**:527, 1931.

reported a case of spongioblastoma multiforme of the intracranial portion of the nerve which grew peripherally and was seen at the disk with the ophthalmoscope.

It is noted that no distinction is made as to the exact point of origin of these tumors in the nerve; we doubt that any of the gliomas arose primarily in the disk within the eye; but this, of course, is a matter of speculation. Should they arise there, however, one would first expect visual symptoms and a tumor visible at the disk long before the development of exophthalmos. In Stallard's case ³⁷ (a primary neurofibroma of the disk) the first symptom was failing vision, the tumor being confined entirely to the disk within the eye.

The most important question, however, is that of treatment. With reference to the meningiomas, the answer is not difficult. These tumors tend to recur; they tend to extend centrally; and no case of distant metastasis is recorded. It would seem, therefore, that the procedure of choice in such cases is craniotomy, followed, if necessary, by exenteration of the orbit or some less radical procedure.

In the second group, that of the glial tumors, the problem is not so simple, and a disagreement exists between us as to the procedure of choice. This type of tumor does not tend to metastasize even locally but rather extends centrally by way of the nerve, contained entirely within the sheath. In at least 1 case ¹⁰ long delayed intracranial involvement is recorded, although in 4 others incomplete removal was not followed by recurrence in periods ranging from seven to seventeen years.

In our second case craniotomy was not done. In Penfield's excellent book,³⁸ Verhoeff made the following statements concerning the gliomas:

Within the nerve stem the growth does not advance by invading the original structure, but by causing the pre-existing neuroglia in the vicinity of the tumor to proliferate and take on the character of the tumor tissue. This would seem to indicate that some substance is produced by the tumor which stimulates the contiguous neuroglia.

And again:

The possible practical importance of this observation lies in its indication that removal of the largest part of the tumor may do away with the assumed stimulating substance and thus prevent further extension of the growth.

Certainly cases such as those previously cited seem to bear out at least the principle of this concept, viz., that simple removal by the anterior approach through the orbit may result in cure. On the other hand, these are isolated instances, and a study of such a large group

37. Stallard, H. B.: Case of Intra-Ocular Neuroma (von Recklinghausen's Disease) of Left Optic Nerve Head, *Brit. J. Ophth.* **22**:11, 1938.

38. Penfield, W.: *Cytology and Cellular Pathology of the Nervous System*, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1035.

of cases as Hudson³ collected shows how inadequately many were studied. On the basis of his 118 collected cases of primary glioma of the optic nerve, the following analysis is made:

Death from meningitis.....	10 cases
Death from recurrence.....	6 cases
Probable recurrence	5 cases
Six year cure.....	6 cases
One year cure.....	34 cases
Patients followed less than one year.....	57

Of the cases in which the patients were followed less than one year, constituting 48 per cent of the cases, we know nothing. And in only 5 per cent of the cases was a six year cure recorded. In the face of these findings, it is impossible for the author on the pathologic side (J. M. W.) to fit into his general concept of tumor growth such a view as is held by Verhoeff,¹¹ viz., that partial removal may be followed by a cure. He believes with Gluck³⁹ that it is a preponderance of the slower growing types that accounts for the relatively long survival after incomplete removal. He therefore advocates primary craniotomy (which he believes is mandatory in those cases in which enlargement of the optic foramen is seen roentgenographically), followed by a second operation from the front for removal of the orbital portion of the tumor which remains. He believes that the problem is one for the neurosurgeon rather than for the ophthalmologist. That there has been a general tendency in this direction since Dandy's paper² in 1922 is noted by the case reports of Mehney¹³ and of Rand and his co-workers.¹⁶ In the latter case the tumor growth extended through the foramen (enlarged by roentgen examination) to a point 1.5 cm. from the chiasma.

That this procedure is too radical may be a just criticism, in view of the long survival in some of the cases cited. The risk is admittedly great. In the 5 cases recorded, including our own case 1, 2 of the patients died of the operation, the growth in 1 case being a primary intracranial tumor (Mehney's case 3). Yet in view of what may be accomplished in some cases, it is thought that the chance of operative fatality is outweighed by the chance of a more likely permanent cure than is offered by the simple removal within the orbit.

SUMMARY

1. Two cases of primary spongioblastoma of the optic nerve are reported, one showing the unusual picture of extension into the optic disk.

39. Gluck, B.: Glioma of the Optic Nerve, *Brit. J. Ophth.* **16**:406, 1932.

2. The need for complete pathologic study of these tumors is stressed, so that by accurate differentiation a more rational prognosis can be made. It is felt that the impregnation methods of Cajal and del Rio-Hortega offer the best means toward this end.

3. Although the operative mortality after craniotomy in the 5 recorded cases is 40 per cent, analysis of the cases reveals a much brighter prognosis, and the neurosurgical approach is advocated by one of us (J. M. W.).

ORTHOPTICS AT THE CROSSROADS

EMANUEL KRIMSKY, M.D.

BROOKLYN

It is only within recent years that the scientific basis for binocular training was advanced and applied by a small group of serious workers, even though some form of exercise for the eyes has been prescribed empirically for many years. Unfortunately, the principles and methods that they fostered won the sympathy of but few ophthalmologists. During the last decade, with its development of instruments for ocular training, there has been on the one hand an unfortunate "groping in the dark" regarding orthoptics and on the other hand an unfriendly skepticism by those too accustomed to established clinical methods, with the result that the favorable aspects of scientifically controlled orthoptic training have been largely obscured. The interest in orthoptic training has not progressed substantially in recent years, and one must reflect as to whether this trial period has been sufficient to evaluate its merits as a therapeutic aid and whether it should be left to proceed on the feeble momentum generated by the sporadic efforts of a few ophthalmologists or be worthy of a more general interest.

A number of British investigators as well as a few in this country have over an extended period carried out studies on various types of binocular imbalance and have discovered distinct benefits in properly selected cases from binocular training carried out in a systematically controlled manner. And yet these reports have not generated interest in controlled orthoptics. I believe that the successful practice of orthoptics presents difficulties that are overwhelming to the average ophthalmologist, and a plan is proposed to ease the responsibilities of the practitioner who would still prefer to supervise personally the management of his cases of binocular imbalance.

The difficulties in controlled orthoptic training are: (1) the matter of expense and (2) the management of the child.

The cost of training based on numerous sittings usually discourages most patients, especially when the length of such treatment cannot be foretold. In my practice I have had the patient purchase or rent a suitable binocular instrument for home use, after a few satisfactory office sittings, in order to save the expense of further frequent visits. Unfortunately, even such an instrument is too expensive for the average patient.

Moreover, the patient has to return every week or two for periodic examinations to determine progress made with binocular or orthoptic

training and for further recommendations as to exercises. This item of expense is not considerable.

Home training requires an intelligent and cooperative parent or guardian who has to understand the purpose of these exercises, the method of exercising on a binocular instrument, how to hold the child's interest, what pictures to use, how long and how often to carry out such training and the necessity of having the child, whenever possible, keep systematic records of his accomplishments; these and many other details demand the cultivation of a specialized interest and training on the part of the ophthalmologist.

The problem of caring for cross eyes and binocular imbalances in preschool children as well as in children in the junior grades cannot be tackled by the ophthalmologist alone. During this formative age period, in which operations are generally avoided, conservative measures, including controlled binocular training, are often advisable in order that possible good may be done to the child. And even when operations are found necessary, these same conservative measures may have to be supplemented in order to maintain the benefits for which the operations were intended. In my opinion, it is only by concerted efforts of both parents and school authorities that such cooperation may be effected. This can be accomplished in two ways: by publicizing the need for early and careful attention to cross eyes and eyestrains and by a simple, practical comprehensive plan for teamwork between the physician and the teacher or the psychologist.

The health teacher or health education department of the school can further the solution of the problem of cross eyes by including among its health gospels such simple teachings as:

1. Early treatment of cross eyes by glasses or by exercises can often correct such a condition. Operation is necessary only when other measures have failed; moreover, it is devoid of danger.

2. Children do not usually outgrow cross eyes.

3. Cross eyes is not a condition to be laughed at but one requiring careful attention to the eyes. (Teachers can do much to counteract the jibes of classmates by keeping photographic posters showing cross eyes before and after treatment.)

4. An eye which has become crossed and is left alone is often likely to turn into a blind eye.

The management of squint is more than a medical or a surgical problem. It is also a psychologic problem,¹ both in regard to the need for a sympathetic understanding of the inferiority complex of the afflicted

1. Krimsky, E.: Psychologic Considerations in the Study of Binocular Function, *Arch. Ophth.* **21**:662-670 (April) 1939.

child and in regard to its correction through nonoperative means, for instance by orthoptic training. However expert the ophthalmologist may be in the surgical or cosmetic correction of squint, comparatively few are at present sufficiently trained or patient enough to administer ocular exercises to the cross-eyed child without assistance. And the assignment of such a child to an orthoptist for supervision and control creates a barrier which neither the ophthalmologist nor the technician desires.

In my opinion, the ophthalmologist who recommends binocular training should either assume personal management of his patient or assign him to another colleague trained in such procedure rather than to a technician. The trained ophthalmologist can evaluate instrumental with clinical findings, whereas the orthoptist thinks primarily of instrumental responses and is likely to attach too much academic importance to detailed readings. In order that orthoptics can be practiced successfully, the ophthalmologist must become as familiar with binocular study as with refraction. He alone should determine whether his patient has an abnormal or a normal retinal correspondence and should be able to ascertain the binocular status accurately. With such a clinical and instrumental record carried out periodically, if necessary, he can better judge the true significance of orthoptic training without actually becoming involved in its detailed application.

With the present economic setup, few children can avail themselves of orthoptic training in a satisfactory manner. In a large city like New York there are only three orthoptic clinics, and these are either unavailable or inaccessible to the large majority of needy children. With such insurmountable difficulties, the cross-eyed child must turn to the school as his haven for binocular training. In recent years the management of other defects of children has been shared by the school system. Among these one may mention: speech difficulties (a psychologic problem), deafness (detected by audiometric tests), cardiac conditions and behavior problems. The training of a child with binocular imbalance includes more than the mere purchase or rental of an instrument; it is a psychologic problem to be studied by one familiar with the peculiar mental makeup of the individual child, and for selected cases of binocular imbalance the school teacher can best attend to such a function without interfering to any appreciable extent with her duties as teacher or with the child's program of work and play. Nor need the teacher be specially trained for such a project, except to have the child report to the ophthalmologist for periodic checkups and for further instructions, which can be written in concise, simple language. The teacher as psychologist may serve as the natural complement to the ophthalmologist as clinician in the correction of squint or binocular imbalance with normal retinal correspondence in selected cases.

CONCLUSIONS

1. The problem of caring for the cross-eyed child from the conservative standpoint is a vast one that cannot be solved by the ophthalmologist alone. It is one that requires an intimate cooperation with the school system. The nursery school or kindergarten provides a sympathetic environment for the young child.

2. Any makeshift or haphazard approach to orthoptic training will justify the criticism of ophthalmologists regarding a method which has till now been looked on with some indifference.

3. It is unfortunate that at the present time but a small number of ophthalmologists are qualified to treat the subject in a critical, analytic manner. It is on the teachings of these men that younger, serious workers will have to depend to spread an approved scientific management of binocular imbalances.

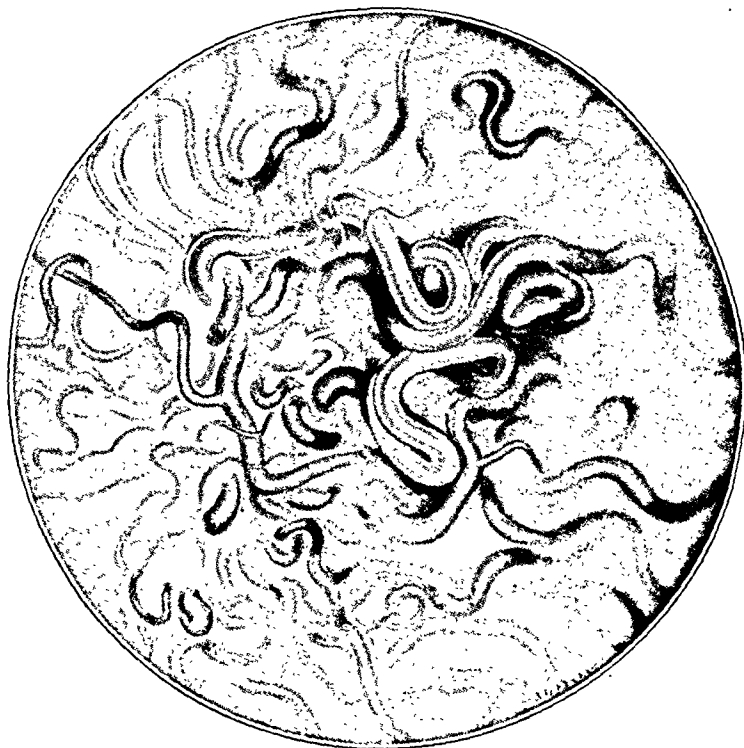
4. Not all ophthalmologists will agree to such a program. As in other fields of human endeavor, one must expect different schools of thought and practice, each with its particular merits; but there must be sufficient representation and serious interest among these competing schools to reduce the incidence of squint and imbalances.

Clinical Notes

ANGIOMATOSIS RETINAE

EDWARD P. RYAN, M.D., NEW YORK

B. T., a 13 year old Jewish boy, was first seen in Dr. Fletcher's clinic at the Manhattan Eye and Ear Hospital on March 4, 1939, with the complaint of failing vision in the left eye for about one year and a half. This impairment of vision was discovered in a routine examination of the eyes at school, but no notice was taken of it by the patient or his family. In my routine examination of the fundi



Drawing of the left fundus of the patient.

an abnormality of the blood vessels was discovered only in the left eye. The vision was 20/20 in the normal right eye. Vision in the left eye was limited to the counting of fingers at 1 foot (33 cm.). Externally every structure was normal in appearance. The pupil reacted to light and in accommodation. The media were clear throughout. The fundus presented an unusual picture of immense, dilated, tortuous and distorted "intestine-like" vessels covering the papilla and the immediate surroundings, particularly the temporal side, as shown in the accompanying drawing. No cystlike formation or structure was found at the periphery of the fundus. There were no exudates, hemorrhages or retinal detachment. Physical examinations, including neurologic, roentgenologic, and otolaryngologic consultations, gave essentially negative results, except for diseased tonsils.

Read before the Section of Ophthalmology of the New York Academy of Medicine, May 15, 1939.

The boy was apparently in good health and still is. His past history and family history were noncontributory. There had been no trauma at any time.

In the case just described the interest is in the tremendous size and the marked tortuosity of the retinal vessels. In view of the rarity of this condition and a diversity of diagnoses, ranging from angiomas of retinae to aneurysma racemosum arteriovenosum, as expressed by several leading ophthalmologists, this case, though titled "angiomas of retinae," might be better classified as a case of abnormality of the retinal vessels, probably of a congenital nature.

KELOID OF THE CONJUNCTIVA

Report of a Case

NORMAN P. SCALA, M.D., WASHINGTON, D. C.

M. A. C., a white woman about 37 years of age, was referred to me by Dr. George W. Creswell on July 26, 1938. She stated that in March 1920, while she was working for the government in Boston, a hemorrhage occurred in her right eye. She was referred by the government authorities to Dr. H. B. C. Riemer, an oculist of Boston, who made a diagnosis of ecchymosis of the bulbar conjunctiva on the temporal side of the right eye.

From that time on, the patient stated, her right eye gave her trouble. She complained of photophobia, severe headaches, the sensation of having a foreign body in her eye and a feeling that her eye was crossed. The left eye was entirely normal. The right eye was normal except for what looked to be a cystoid degeneration of the conjunctiva on the temporal side just posterior to the insertion of the external rectus muscle.

When the patient was sent to me, it was thought that she had a foreign body in her eye. However, a thorough examination did not reveal the presence of one.

Several attempts were made to remedy the condition by superficial removal of the tissue, but after each attempt it only became more aggravated, and the mass increased in size after each operation.

It therefore became necessary to hospitalize the patient and remove the affected tissue down to the sclera, including some of the muscle fibers. The tissue was removed well outside the affected area; the denuded surface was covered with healthy conjunctiva from above and below, and four interrupted sutures were inserted. The patient was hospitalized from September 12 to 26 and was unable to return to work until October 17.

The tissue was sent for examination to Col. J. E. Ash,¹ of the Army Medical Museum, who made the following report:

"The specimen from your patient, M. A. C., our accession no. 63668, is a minute piece of firm fibrous tissue showing nothing significant grossly.

"Microscopically the specimen is partially covered by conjunctival epithelium, just beneath which there are foci of edema and congestion but very little exudate. The bulk of the mass consists of dense collagenous bundles containing few elongated and narrow nuclei, groups of blood vessels which have the appearance of being

1. Curator, Army Medical Museum, Washington, D. C.

the remnants of granulation tissue, several thick-walled and older vessels and some small mononuclear exudate scattered about the younger blood vessels. I can see no neoplasm and would interpret the picture as quite hyperplastic scar tissue showing a keloid tendency."

COMMENT

Since the removal of the tumor formation and the healing of the wound, the patient stated that all of her symptoms have abated and that she is able to continue her work with comfort and without glasses. It is probable that this condition started in 1920 when she had the hemorrhage, which was probably due to the strain the eye was under from the great amount of work she was compelled to do and the long hours required to do it. The symptoms complained of afterward were, no doubt, the result of scar tissue which formed in the region of the external rectus muscle and involved some of its fibers, thereby causing a limitation of the movement of the muscle. The removal of this mass together with the muscle fibers gave the eye more play and resulted in an abatement of the symptoms, for which the patient was grateful.

SUMMARY

A case of keloid of the conjunctiva with marked subjective symptoms is reported. Clinically the picture was that of cystic degeneration of the conjunctiva and was indistinguishable from that of the more common type of such degeneration.

From a review of the literature, it is evident that a keloid of the conjunctiva is a rare condition, as this is the first case that could be found that has been reported. There has been reported, however, to the Eye Registry, Army Medical Museum (accession no. 38976) an instance of keloid of cornea² by Dr. Henry C. Smith, of Nashville, Tenn., and I understand that he is preparing a report of this case for publication.

2. Smith, H. C.: Keloid of Cornea, to be published.

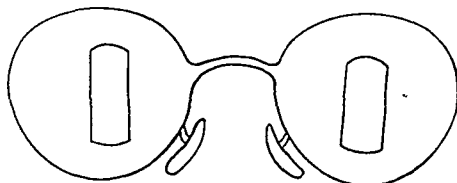
SPECTACLE LENSES FOR USE DURING OPERATIONS

GEORGE M. RICHARDS, M.D., STONY POINT, N. Y.

To overcome some of the disadvantages presented by the use of the Beebe loupe and its modification during an operation, especially when the operator is presbyopic or desires a comfortable working focus of 15 to 20 cm., the following bifocal lens was designed.

The essential advantage of this lens is that a working area is afforded in the upper field as well as in the lower, and at the same time free lateral vision is permitted. To accomplish this a segment 10 by 24 mm. is placed almost vertically near the middle of an ordinary spectacle lens and equidistant from the top and the bottom of the lens. The upper pole of each segment is tilted templeward 1 mm. to allow for separation of the visual axes in the upper fields. The segments are

centered for the operator's pupillary distance when his eyes are focusing at 15 to 20 cm. A 1 degree prism, base in, is ground in each segment to relieve some of the excessive convergence at this short focus. The lens on which the bifocal segment is placed can have the operator's distance correction or an intermediate correction to focus at about 60 cm., which is convenient for reaching and selecting instruments. The strength



Spectacle lenses for use during an operation.

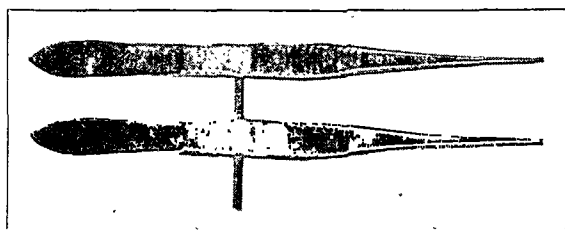
of the bifocal lens will average near a $+3.00$ addition to the intermediate correction or a $+4.00$ addition to the distance correction. The segments are cemented in place. If a pad-bridge frame is used, large (jumbo) pads are recommended to distribute better the extra weight of the frame and lenses.

This design, shown in the accompanying sketch, is submitted in the hope that it will help others who have had similar difficulties.

BIPOLAR FIXATION

ROBERT R. BLONDIS, M.D., CLEVELAND

The desirability of bipolar fixation of the globe during surgical procedures has frequently been mentioned. This is usually obtained with the aid of an assistant. The forceps pictured here obviate the necessity for an extra hand in the operative field, since pressure on



Bipolar fixation forceps.

one forceps operates also the other and gives satisfactory bipolar fixation. The instrument consists of two ordinary fixation forceps, which can be locking or nonlocking in type, on a sliding bar. The distance between them can be varied and exactly maintained. A measured scale on the bars can be used for the exact resection of muscle or other tissue. More individual forceps can be placed on the double bars and used to approximate incisions in the skin and other structures evenly.

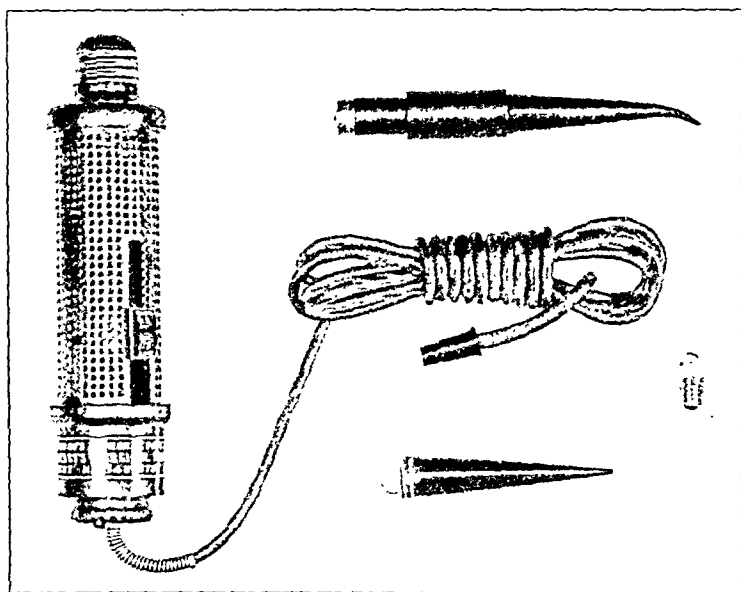
OCULAR TRANSILLUMINATORS MADE OF LUCITE

HAROLD M. BLOCK, M.D., DALLAS, TEXAS

The transilluminators described here are not new in the field of ophthalmic instruments. They are a modification of those made by a European manufacturer of instruments.

Since lucite¹ has optical properties similar to those of quartz, it lends itself well to the transmission of light. It has the disadvantage, however, of being destroyed or injured by high temperatures and various chemicals, but it is easily molded and worked with.

The transilluminators described here are each approximately 3 inches (7.6 cm.) long and taper from 13 mm. at their base, where light is admitted, to 1.5 mm. at its point of exit. One of the transilluminators is



Ocular transilluminators made of lucite.

straight; the other has a slight curve, the light emerging at an angle of approximately 45 degrees. The straight transilluminator is adapted for examination of the more anterior parts of the globe, and the curved one is adapted for inspection of the deeper recesses in the cul-de-sacs. With the curved type, light is thrown toward the interior of the globe through the sclera, which of course is impossible with a straight transilluminator. The curved transilluminator is silvered on its outer surface and then opaqued, but the straight one is only opaqued.

The light source is a magnifying globe of the flashlight type, with a double filament. It operates on a variable current of from 6 to 7 volts.

From the Department of Ophthalmology, Baylor University College of Medicine and Baylor University Hospital.

Mr. Don Cameron, of the Cameron Surgical Specialty Company, lent technical aid.

1. Lucite is a du Pont product (methyl methacrylate).

This globe gives out an intense beam of light, which is picked up and concentrated through the small opening at the exit of the transilluminator and measures $2\frac{1}{2}$ foot candles on a current of 7 volts. A rheostat is available so that the intensity of the illumination can be varied.

Owing to the physical properties of lucite and the opaquing process, the solutions for sterilization are limited. These four may be used: potassium mercuric iodide (Parke, Davis & Co., germicidal disks), 1 tablet in 4 ounces (120 cc.) of water, freshly prepared; mercury bichloride, 1:400 solution; hexylresorcinol, 1:1,000 solution, and a solution of merthiolate. I have used hexylresorcinol, as I do not believe it will damage the metal parts of the transilluminator as do the metallic salts or iodine. However, the instruments are rarely, if ever, used in actively infected eyes, and sterilization under these circumstances is not as difficult.

My own experience with these instruments has proved highly satisfactory, and I am well pleased. The only disadvantage is that the light in the handle gets warm after three to five minutes' usage, but this is not a serious drawback, since the tip of the transilluminator, that part in contact with the eyeball, remains cool. A local anesthetic of one of the cocaine substitutes is sufficient when these instruments are employed. Patients react well, as there is no pain, heat, glare or pressure.

There are already several ophthalmic transilluminators on the market, but in designing these I paid particular attention to the exit point of light. If a tumor is small, say 1 to 2 mm. in size, and the opening larger than the tumor itself, the transmission of light will not be inhibited. Since the point of exit of light in these transilluminators is about 1.5 mm., I feel that in event of a small tumor or foreign body the transmission of light will be inhibited, making the contrast more noticeable. With the tip of the opaqued transilluminator against the globe, there are no blinding or confusing rays of light in a completely dark room. The observer, then, can get the maximum contrast from all accessible areas of the sclera.

513-514 Medical Arts Building.

News and Notes

EDITED BY W. L. BENEDICT

UNIVERSITY NEWS

Postgraduate Course in Aviation Ophthalmology and Aviation Medicine.—A postgraduate course in aviation ophthalmology and aviation medicine for graduates in medicine will be given by the George Washington University School of Medicine, Washington, D. C., from April 1 to 6 inclusive. This course is part of the postgraduate course in ophthalmology given from March 19 to 30 inclusive.

Course I is a special practical course in ocular surgery, pathology and orthoptics, limited to 25 participants, March 19 to 23 inclusive; course II, an intensive postgraduate course in ophthalmology, March 25 to 30 inclusive, and course III, a postgraduate course in aviation ophthalmology and aviation medicine, April 1 to 6 inclusive.

Among the guest lecturers are 17 officers of the Medical Corps of the U. S. Army and the U. S. Navy and Civil Aeronautics Authority. Credit will be given by the War Department and the Navy Department to those officers in the Medical Reserve Corps of the U. S. Army and the U. S. Navy who take course III. The fee for this course will be \$40. The course will be given under the direction of Dr. W. Thornwall Davis, professor of ophthalmology at George Washington Medical School, assisted by the members of the staff of the department of ophthalmology.

Course in Visual Optics and Physiology.—The Harvard Medical School will give a course in visual optics and physiology from July 1 to July 27, 1940. This course, conducted by Drs. Ludvigh, Cogan and Easton with the assistance of other members of the staff, will be an all day course given six days a week during the month of July. Lectures, laboratory work and discussion will occupy seven hours a day, and outside reading will be assigned covering articles on special subjects in the modern American and English literature. The first two weeks of the course will cover approximately the range of material given in Duke-Elder's "Textbook of Ophthalmology" (vol. 1, pp. 483 to 1086). Subsequently, a treatment of more advanced topics not thoroughly discussed in the average English or American textbook will be undertaken.

PERSONAL

Dr. R. Townley Paton has been appointed professor of ophthalmology and head of the department of ophthalmology at the New York Medical College. Dr. Edwin S. Munson, who preceded Dr. Paton, has been retired for three years. The old New York Ophthalmic Hospital has been recently merged with the college. Dr. Paton will retain his position as surgeon at the Manhattan Eye and Ear Infirmary but will devote most of his time to the reorganization of the department of ophthalmology at the New York Medical College.

Dr. Charles M. Swab has been appointed director of the department of ophthalmology at the Creighton University School of Medicine. Dr.

Swab graduated from Creighton University in 1916 and received an M. S. degree in medicine from the University of Pennsylvania in 1934. He has been associated with the department of ophthalmology in the School of Medicine and the Creighton Memorial St. Joseph's Hospital in the capacity of associate professor since 1932.

Dr. Louis B. Bushman, who has been director of the department since 1919, has been made professor emeritus.

SOCIETY NEWS

Oxford Ophthalmological Congress.—The Oxford Ophthalmological Congress will be held at Oxford, England, from July 4 to 6, 1940. Prof. W. E. Le Gros Clark will deliver the Doyne Memorial Lecture, and there will be a discussion on "Emergencies and Complications of the Operation for Cataract."

Ophthalmological Society of the United Kingdom.—The Annual Congress of the Ophthalmological Society of the United Kingdom will be held in London, England, at the Royal Society of Medicine on April 25 and 26, 1940.

The subject for discussion will be "The Choice of Operation for Glaucoma." The discussion will be opened by Prof. A. J. Ballantyne.

Members wishing to read papers are asked to send their titles to Mr. J. H. Doggart, 49 Wimpole Street, London, W. I., as soon as possible. Abstracts of these papers should be sent not later than March 23, 1940, to be printed for circulation at the congress.

GENERAL

A World Assessment of Blindness.—"The International Association for the Prevention of Blindness undertook in the past years an inquiry into the incidence of blindness throughout the many countries of the world, and of the regulations in force in those countries for the prevention of blindness. This report has now been published in French (*Internat. Ophth. Cong. [Concilium ophth., 1937] 7: 1-310, 1938*). It is an important piece of work of 310 pages, and full of information collected by the International Association. No fewer than thirty-seven countries responded to the inquiry. It is of much interest to compare the reports and to note the differences shown in them, not only in the variation in the main causes of blindness but also in the number of the blind in relation to the populations. In the East the figures are estimates, and they are high owing mostly to the widespread incidence of trachoma. In some of the Western countries the figures seem to be contradictory, for there is recorded a higher proportion of blindness in some of those countries where we know most is done, and done successfully, for the prevention of blindness. The reason for that apparent contradiction is that in these countries, like England, blindness is measured by its economic disability and not by a total absence of sight. The report is a valuable document and the Egyptian publication is well done."

Correspondence

IS THE ONSET OF INTERSTITIAL KERATITIS RELATED TO RIBOFLAVIN DEFICIENCY?

To the Editor:—A year ago there appeared, almost simultaneously, an article by Bessey and Wolbach (*J. Exper. Med.* 69: 1 [Jan.] 1939) and one by us (*ARCH. OPHTH.* 21: 315 [Feb.] 1939) relating to observations on the cornea of rats fed diets deficient in riboflavin. The vascularization observed was depicted by each of us, the article by Bessey and Wolbach containing some excellent drawings, and our article, a photograph of a cornea the vessels of which had been injected with india ink. Bessey and Wolbach stated: "Vascularization of the cornea is an early and constant phenomenon in albino rats in riboflavin deficiency. It precedes all other demonstrable lesions of the deficiency." We stated: "From our experience with the diet deficient in riboflavin, the most consistent ocular change was the appearance of corneal vascularization. . . . After riboflavin was added, the most uniform effect was the remission of vascularization."

On Dec. 6, 1938, one of us (L. V. J.) discussed with Dr. Arnold Knapp the results of some six months' experience with riboflavin as used for the relief of human vascularization of the cornea, observations having been made on some 40 patients with all sorts of vascularization, from pannus of trachoma to interstitial keratitis. By that time we had observed that rosacea keratitis responded most satisfactorily and interstitial keratitis and the pannus of trachoma most slowly. These studies were made with natural riboflavin, crystallized from milk whey (supplied by general biochemicals division of the S. M. A. Corporation), a small quantity of which was supplied to Dr. Knapp for his clinical use, limited only because of the great expense of its preparation. At this time we considered the minimal daily therapeutic amount to be 3 mg. This is the approximate riboflavin equivalent of 3 quarts (2.8 liters) of milk. Since at that time (and we believe this is still true at the present) commercial vitamin products contained around 50 micrograms of riboflavin, or the equivalent of a twentieth of a quart of milk, per unit of therapy, we felt it wise not to announce our satisfaction with riboflavin, knowing that the result would be a series of disappointing clinical trials made with advertised vitamin products which were almost void of the riboflavin, even though they contained adequate amounts of vitamins A, B₁, D or C.

Ophthalmologists will be disappointed with clinical results if, after reading the accounts widely distributed through the newspapers of all cities, they attempt to give relief to their patients by the use of preparations containing the riboflavin equivalent of a twentieth of a quart of milk! They will be especially disappointed if they take literally the paragraph as it appeared in a copyrighted article by Science Service, as it appeared in the *Cleveland Press* of Feb. 3, 1940 (reported to be

released by Drs. H. D. Kruse, V. P. Sydenstricker, W. H. Sebrell and H. M. Cleckley and announced through the United States Public Health Service).

It was stated: "Babies born with syphilis, formerly thought to be a cause of the eye disease, can now have their eyes cured." These conclusions, the article reports, are based on the observations of 2 cases! Until commercial products contain riboflavin equivalent to at least a glass of milk per unit of therapy, we advise that patients drink milk.

One of us (L. V. J.) has used riboflavin for as long as a year for some patients with active and inactive interstitial keratitis and has observed no such dramatic disappearance of blood vessels. It is his opinion, however, that there is a definite value, since a far greater number of blood vessels than would be ordinarily expected are eventually empty of blood cells and with the slit lamp are seen as empty channels. It appears that from 6 to 9 mg. daily, taken orally, is adequate, and no additional benefits resulted from the intravenous injection of 15 mg. daily.

In cooperation with the department of dermatology of Western Reserve University School of Medicine during the fall of 1938, we attempted to produce interstitial keratitis in rabbits which were infected with syphilis by maintaining them on a diet void of riboflavin, but no vascularization or evidence of inflammation occurred; nor was it possible to secure a litter of rabbits from a doe so infected and maintained on a diet deficient in riboflavin.

It is of chronologic interest only that Dr. Paul György (on whose suggestion the first animal experiments were carried out and who has cooperated as vitamin advisor throughout the complete study) was given permission to discuss our findings concerning vascularization of the human cornea at (1) the Academy of Medicine of Cleveland in a post-graduate lecture delivered on Feb. 13, 1939 (mimeographed copies of which were widely distributed at 10 cents each) and at (2) a meeting of the Milwaukee Pediatric Society in April 1939.

LORAND V. JOHNSON, M.D., Cleveland.

ROBERT E. ECKARDT, M.S., Cleveland.

Obituaries

HARVEY CUSHING, M.D.

1869-1939

HIS CONTRIBUTIONS TO OPHTHALMOLOGY

On April 8, 1869, in Cleveland, Harvey Williams Cushing was born, and on Oct. 7, 1939, in New Haven, Conn., he died, in the seventy-first year of his life. He was the founder of a school of neurosurgery, being one of the few men ever to have founded a distinct school of surgery. His pupils assert that his name will go down in history as that of one of the greatest surgeons of all time. He was a teacher, author, biographer and philosopher. In the six months since his death, eulogies, tributes, appreciations and editorials, in medical and lay papers, have set forth the fruits of his stupendous industry and marvelous technic. Recorded again and again are the customary dates and details. His father and his father's father and his great-grandfather were physicians; so medicine "ran in his blood." Of pure New England stock, transplanted beyond the Alleghanies, it was natural, as so often happens in the unmixed race of New England, that he inherited family traits and the family pursuit. In order to follow him from place to place as he went on his course through life, a few chronologic facts are here recorded. He was graduated from Yale in 1891 with the degree of Bachelor of Arts and from Harvard in 1895 with the degree of Doctor of Medicine and Master of Arts. As to his surgical homes and positions, he was house officer at the Massachusetts General Hospital from 1895 to 1896. The year 1896 marked the beginning of his connection with the Johns Hopkins Hospital and Medical School. One year of this period, which he referred to as the happiest year of his life, he spent in research work abroad, making the trip over the Atlantic with William Osler. He worked under the surgeon Theodor Kocher and the pathologist Hugo Kronecker, both men of great renown, in Berne, Switzerland. He ended the year with the famous pathologist Sir Charles S. Sherrington in Liverpool, England. He had tried to work with Sir Victor Horsley in London, England, but this famous neural surgeon was far too busy to attempt to teach him. Returning to Johns Hopkins, he was promoted from the rank of assistant resident surgeon, and on up until he was made associate in surgery in 1902 and associate professor of surgery in 1903. He occupied these two positions until 1912, when, after sixteen

Read before the Section of Ophthalmology of the New York Academy of Medicine, Feb. 19, 1940.

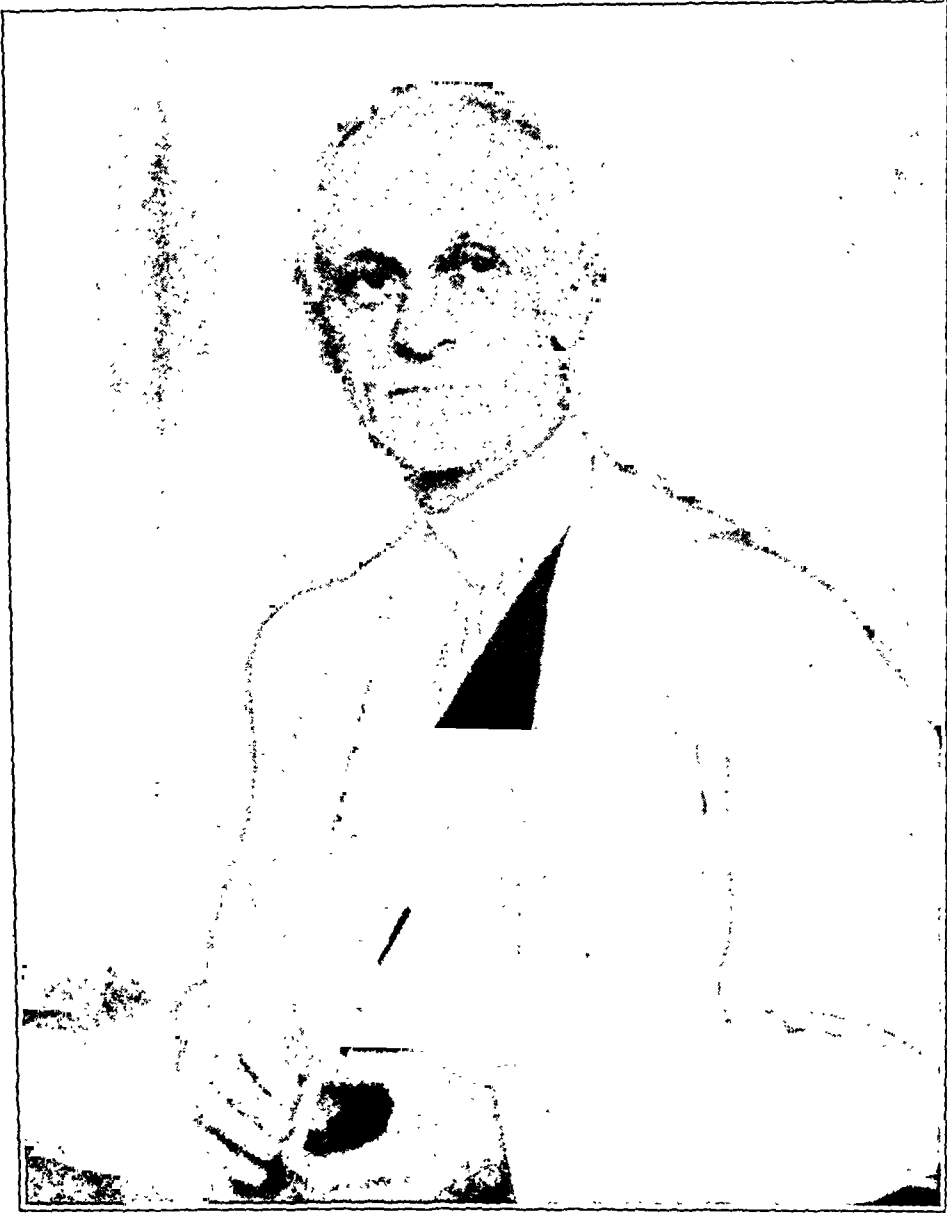
years of service, he returned to his medical alma mater, Harvard, and entered on the duties of surgeon in chief of the newly built Peter Bent Brigham Hospital and professor of surgery in the medical school. In 1932, this being the year set by himself for retirement from active surgical work, he became emeritus in both institutions.

There was an interruption in his Harvard connection from 1917 to 1919, when, with the rank of colonel, he served in the World War as director of the United States Army's Base Hospital No. 5 in France. In 1918 he was appointed senior consultant in neurologic surgery in the American Expeditionary Forces.

In 1933 he was persuaded to return to Yale, from which he had been graduated forty-two years before, to become Sterling professor of neurology. With him went his collection of 2,000 tumors of the brain, with photostatic copies of their histories, and his vast library of medical books, believed of its kind never to have been surpassed by any ever held in private hands. In 1937 he became emeritus. At the time of his death he was director of studies in the history of medicine at Yale. Thus it is seen that his footsteps ended, after forty-eight years, where they began in his preparatory work for medicine. How many times for brief periods was he called away from his surgical homes at Johns Hopkins, Harvard and Yale to deliver lectures and orations or to receive honors and degrees in institutions of learning in this country and abroad!

It was in 1901 that he delivered the Mütter lecture in Philadelphia, at the early age of 32. This lecture was entitled "Some Experimental and Clinical Observations Concerning States of Increased Intracranial Tension." It was based on experimental work that he had done under Kocher in Berne. The lecture was followed two years later, in 1903, by a paper entitled "On Routine Determinations of Arterial Tension in Operating Room and Clinic," read at the Boston Medical Library. This paper marks the beginning of blood pressure determination in this country. Cushing was now launched on his remarkable career as a formal lecturer and deliverer of orations. The topic always fitted the occasion, and many of the papers so presented were of profound scientific importance. His addresses were by no means confined to medical subjects, but, as the event required, he delivered lectures on the history of an institution whose guest he happened to be or on the life and times of some great medical personage. It was said that he always had the "feel" of any institution that he visited and that he made many inquiries about its history and often put to shame those connected with it through knowledge of its traditions greater than theirs. His vacations and peregrinations were different from the standards set by most medical men in that they were given up to lectures or to visits to distant clinics in an insatiable search for knowledge.

In England, in the summer of 1920, unheralded he appeared at the Oxford Ophthalmological Congress. The presence of so distinguished a guest having been made known to the master, he was called on to take part in a discussion on perimetry, to the opening of which he had been listening. He confessed that he had come "to see what an ophthal-



HARVEY CUSHING, M.D.

1869-1939

mologic congress was like" and that he had been most interested in a discussion on the humanities that had preceded the principal subject. He admitted that he had been taking notes and that he had remained longer than he had intended because of his great interest in perimetry as an aid in his neurosurgical work.

In the course of time the figure of this small, alert, wiry man, with a deep mellow voice, became a familiar one in the deliberations of two generations of surgeons, scientists, philosophers, historians and librarians throughout the length and breadth of this country and in Canada and Great Britain.

In addition to the preparation for elaborate lectures and orations and in spite of the precious time consumed in delivering them in distant parts, the heavier work on books and monographs was pursued with a vigor that never abated. The mere enumeration of their titles and dates is as a diary of the days of his years, reflecting the limitless breadth of his activities. In 1912 there came out "Pituitary Body"; in 1917, "Tumors of Nervus Acusticus" and "Story of Base Hospital No. 5"; in 1925, "Life of Sir William Osler"; in 1926, "Classification of Gliomas and Studies in Intracranial Physiology"; in 1927, "Pathological Findings in Acromegaly"; in 1928, "Tumors Arising from Blood Vessels" and "Consecratio medici"; in 1932, "Intracranial Tumors" and "Pituitary Body and Hypothalamus"; in 1936, "From a Surgeon's Journal," and finally, in 1938, "Meningiomas" (with L. Eisenhardt), the largest of his monographs and regarded as his best. What nuggets of purest gold did this master surgeon strew along the pathway of the last six and twenty years of his life, not the least being a description of a syndrome associated with basophilic adenomas of the pituitary gland, now known as "Cushing's disease." Herein lies his noble contribution to endocrinology. Many of his works are replete with references to the eye and are rich in illustrations of the effects of tumors on the visual fields. Every ophthalmologist should have Cushing's volumes on the shelves of his library. They would prove a constant source of information and inspiration.

The sixtieth anniversary of his birth, in 1929, did not pass unheeded. A special number of the *Archives of Surgery* was issued in his honor, containing eighty-two papers. The contributors were former pupils, some of whom by this time had risen to positions of prominence in medicine and surgery. What they had learned from him had come through his example and precept and the good old established method of apprenticeship—"The hippocratic method," as he termed it. Cushing was called "the surgeon of the written word," because from his college days he was in the habit of taking copious notes, embellishing them with accurate and artistic free hand sketches.

The basis of his surgical technic lay in a thorough knowledge of the pure anatomy of the field in which he worked. Experiments, begun in the earliest years of his medical career and never discontinued, contributed to his unrivaled perfection as a neural physiologist. He belonged to the "Halsted school" of surgery, which teaches that deliberation and precision, with perfect hemostasis, lend efficiency and dignity to an

operation. Hitherto, in the preanesthetic days, swiftness was the measure of the ability of a surgeon. This was particularly true of the English surgeon Sir. Astley Cooper, who like Cushing was a prolific writer and a world renowned operator and teacher. But with Cushing, it was far otherwise. His operations on the brain were timed by hours, not minutes. He seldom operated on any other part of the body, although he was surgeon in chief at Harvard. He did not instruct during an operation, but he was a performer, soul and body entering into the task. He lacked the gifts of moving oratory and extemporaneous speaking. Exquisite lightness and dexterity characterized his handling of all tissues. Probably no surgeon ever lived who felt the sanctity of the living normal cell as deeply as he or tried more conscientiously to protect it from unnecessary injury. From all accounts it must have been with him, as his friend John Chalmers Da Costa wrote of Philadelphia's famous surgeon Pancoast, "he had a hand as light as a floating perfume and an eye as quick as a flashing sunbeam."

It is an old aphorism that there is no great reward without great labor. This was true of Cushing. He wrought in hours stolen from family and friends and from periods of much needed rest. He knew what it was to suffer in his own person, for, known to but few, since the World War he often labored under pain due to peripheral vascular disease that came on during his arduous duties in France. Neither was he spared in his surgical life, as he confessed in retrospect "sudden harrowing tragedies which serve to unnerve a conscientious surgeon."

The first signal honor to be conferred on him came in 1913, the year after the publication of "Pituitary Body," in the form of the degree Master of Arts (honorary) from Yale. This same year, as a further indication of the eminence he had attained, he was called to be orator in surgery at the seventeenth International Congress of Medicine at London. During his stay in England he was elected a fellow (honorary) of the Royal College of Surgeons. Degrees, fellowships, titles and honorary memberships followed fast on one another's heels. Particularly flattering was the award of the Pulitzer Prize in Letters of Columbia University in 1926, in recognition of his "Life of Sir William Osler," on which he labored with an amazing amount of collected details for five years. This put the seal of approval on his humanistic accomplishments. Whether in peace or in war, whatever he touched he adorned. Whenever he was asked to go a mile, he went twain! He was made Officer of the Legion of Honor, and he was as worthy of this distinction as Larrey, Napoleon's military surgeon, of whom it is written: "He followed Napoleon with love that never failed, with constancy that never faltered, and gave all his best skill to friend and foe as duty called." From his own country Cushing received the United States Distinguished

Service Medal, and in England he was made Companion of the Bath (military). But the crowning recognition of his merit, in comparison to which all others were as stepping stones, came in 1938, when he crossed the ocean to receive from Oxford University the degree Doctor of Science, "honoris causa," an unprecedented honor for an American physician. To felicitate him on this high occasion, there assembled at Oxford a notable gathering of his former pupils and of colleagues from many countries of Europe. On a Sunday morning he held at Radcliffe Hospital, Oxford, a clinic that those who attended said they would never forget as long as they remembered anything. Only a few weeks before his death there came a cablegram from the Council of the Royal College of Surgeons, London, nominating him for honorary fellowship, to which he cabled back, "Refusal by a surgeon of such an unusual honor inconceivable."

A list of all degrees and honors conferred on him runs into pages. Two there are that concern ophthalmologists particularly. One is the award to Cushing of the Knapp Prize in Ophthalmology in 1928, for the paper entitled "Meningiomas Arising from the Tuberculum Sellae with the Syndrome of Primary Optic Atrophy and Bitemporal Field Defects Combined with a Normal Sella Turcica in a Middle-Aged Person (with L. Eisenhardt)." This, one of Cushing's most informative papers, was read before the Section on Ophthalmology of the American Medical Association at Minneapolis in 1928. The other honor referred to is his election to honorary membership in the American Ophthalmological Society in 1938 in recognition of all that he had done to enlighten, elevate and broaden this special branch of surgery. But if he had helped ophthalmologists, they also had been of help to him. Hear what he modestly said at his seventieth birthday party, given by the Harvey Cushing Society at New Haven, April 8, 1939: "I have spent my neurosurgical life leaning heavily on others: on Percival Bailey and his pupil Louise [Eisenhardt] in histopathology; on Clifford Walker for keeping our perimetry reasonably accurate by periodically coming on from the West Coast to check our procedures; on Larry Reynolds and Merrill Sosman for their unfailing interest in the roentgenological features of the work; on John Fulton for keeping us attuned in these later years to its physiological aspects; and on many more of you for assistance at the operating table, more particularly on Gil Horrax, who for so long a time carried the brunt of the load." Then he referred to the faces that he sorely missed, and he named several as being "doubly dead in that they died so young." Perhaps he had also in mind George Derby, professor of ophthalmology at Harvard, with whom he worked in clinical collaboration and whose memoir he wrote in a touching but searching vein. He could have gone further and declared with Cooper that much of his success depended on his zeal and industry, for

which he could take no credit, as it was given to him from above. Among the spontaneous tributes that he heard at this memorable party were letters from patients bespeaking their inexpressible thankfulness for restoration of their sight. He always asserted that this result was the crux of his operations.

The acknowledgment of his debt to ophthalmologists on his last birthday was not the first. At the meeting of the Oxford Ophthalmological Society, already alluded to, in unpremeditated remarks he referred to Clifford Walker, then on his staff at Harvard, as "a mathematical genius of the Helmholtzian order," who, by refining the technic of perimetry, had made it possible to arrive at a "precocious diagnosis" of very early pressure symptoms, especially of changes in the chiasm due to pressure by pituitary lesions. Cushing and Walker strove to create a standard method for the taking of visual fields, which could be passed on to those interested in ophthalmology "but who in the matter of practice stood only on the threshold of the specialty." To fill this need, Walker devised the special chart and disks and the special technic for the quantitative taking of fields now in use in neurologic clinics. Said Cushing, more important than the instrument is the perimetrist, and he stressed the importance of the peripheral field to the neurosurgeon.

Further elucidation of what came to be known as "Cushing's syndrome" was contained in a paper entitled, "The Chiasmal Syndrome of Primary Optic Atrophy and Bitemporal Field Defects in Adult Patients with a Normal Sella Turcica," read at the thirteenth International Ophthalmologic Congress, at Amsterdam, Netherlands, in 1929. This served to strengthen the link between neurosurgical and ophthalmologic surgeons. The latter were brought to realize the importance of early and accurate perimetry, as patients with tumor of the brain are prone to consult them first.

Cushing more than once referred to the ophthalmologic surgeon as being the oldest of the surgical specialists and to the neurosurgeon as being the youngest. He observed that the neurologists had begun to imitate the ophthalmologists by becoming surgically minded. He spoke of the optic chiasm as constituting "the cross-roads" where ophthalmologists and neurologists have a common place of meeting. He explained that the modern neurosurgeon has come to deal largely with lesions that mechanically affect the intracranial part of the apparatus of vision, whereas the ophthalmologic surgeon limits his operative procedures to the intraorbital portion. He settled the mooted question as to whether meningiomas in the sheath of the optic nerve have an intracranial or an extracranial origin. He proved that they may arise primarily in either location and may progress either way through the optic foramen. He believed in "seeing all round" a tumor. He pointed out that in orbital operations ophthalmologists were in the habit of

attacking a mass from in front or from the side, but he was sure that many of them should be approached to "the windward," that is, from the rear and above, and that only a neurologic surgeon is capable of doing this. He maintained that what is known as "choked disk" is a mechanical rather than an inflammatory process in the vast majority of cases, else vision would not be restored so soon after the mere removal of a tumor. He emphasized that tumors of the brain are too heterogeneous to be grouped together and that they differ not only in type but in sites of predilection and in life history and prognosis. Through his endeavors, that which was once considered hopeless and inoperable in the brain may now be operated on with a degree of security and success to the patient comparable to that given him in an abdominal operation. Happily gone now is the century-old teaching that the cardinal symptoms of a tumor of the brain are headache, vomiting and choked disk.

Admiration, praise and gratitude are written into these lines, which only hint at his achievements. But what was the personal attitude of this man, who was aristocratic and reputed by some to be austere, toward ophthalmologists? The answer may be found in a letter that he wrote to the secretary of the American Ophthalmological Society in 1938, in which, after expressing his deep appreciation and gratitude for the compliment paid him on his election to honorary membership to which he had always aspired, he stated: "My special field of work took me into ophthalmology no less than into neurology, and I have always looked up to the ophthalmologists not only as having been the first surgical specialists but as representing in their persons the elite of the profession."

It is a duty and an honor that this section discharges in pausing to reflect on the life of a peerless surgeon now removed from earthly scenes.

BERNARD SAMUELS.

Obituaries

PATRICK CHALMERS JAMESON, M.D.

1867-1939

Dr. Patrick Chalmers Jameson was born in Kircaldy, Scotland, on Sept. 22, 1867 and died at the Long Island College Hospital on Oct. 27, 1939, after a short illness. He was descended from old Scotch ancestry. His father, Charles Jameson, was a preacher in the Presbyterian church. His mother, Grace Chalmers, was a niece of Dr. Thomas Chalmers, one of Scotland's renowned ministers.

He was the youngest of seven children and obtained his early schooling in Edinburgh, where he also attended the Merchiston Castle School. To recuperate from his strenuous training, and having a great desire to see the world, he came to America and spent four years on various ranches in the Southwest. In Texas he acquired a ranch of his own. Here he developed a strong physique but missed intellectual companionship, and this lack of mental stimulation prompted him to return to Scotland.

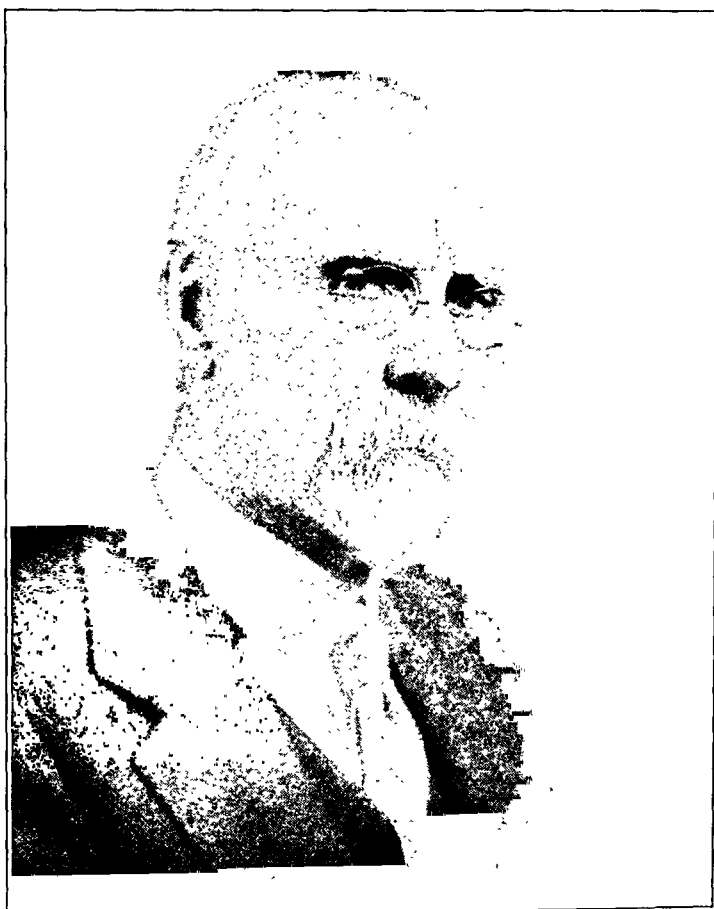
Having set his heart on a profession, he decided to study medicine. He returned to America and entered the Long Island College Hospital in Brooklyn, from which he graduated in 1892. He was influenced in the choice of this school by his great admiration of Prof. Alexander J. C. Skene, who then held the chair of gynecology in that institution. Dr. Skene was also a Scotchman, and the racial ties were strong and always remained so with Dr. Jameson.

He served his internship at the Brooklyn Hospital, and there he became especially interested in surgery and pathologic research, which he continued at the Hoagland Laboratory with Dr. Archibald Murray and the late Dr. Randolph. Later, becoming interested in ophthalmology, he attended the clinics of Dr. John Weeks and the late Dr. Herman Knapp. He also spent several summers in some of the European clinics, especially at Bonn, Utrecht and Wiesbaden with Professor Saemish, Professor Snellen and Dr. Pagenstecher.

His association with the Brooklyn Eye and Ear Hospital began in 1896, under the late Dr. Arthur Mathewson; on the latter's retirement, he took over his private practice. He served the Brooklyn Eye and Ear Hospital for many years: for twenty-six years as a surgeon and for his last seven years in the capacity of senior surgeon. As an active

member of the board of directors and its secretary since 1927, he was most instrumental in obtaining the new hospital building, which was opened in 1930. At the time of his death he was also consulting ophthalmic surgeon to the Brooklyn Hospital, St. Johns Hospital and the Caledonian Hospital.

Dr. Jameson enjoyed a large private practice. His patients were most devoted and loyal to him.



PATRICK CHALMERS JAMESON, M.D.

1867-1939

He was a member of the American Medical Association, the New York state and county medical societies, the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology, the New York Ophthalmological Society, the Brooklyn Ophthalmological Society, the Associated Physicians of Long Island and the Practitioners Club. He was a fellow of the American College of Surgeons and belonged to the Alumni Society of the Brooklyn Hospital.

Dr. Jameson contributed generously to the various journals, and with a special aptitude toward surgery, he devised and improved several operative procedures. Some of his papers are:

Observations on the Prophylaxis of Ophthalmia Neonatorum, 1899.

Injury of an Eye, Necessitating Enucleation Twenty Years Later, *Brooklyn Medical Journal*, 1900.

The Bacteriological Element in the Etiology of Acute Catarrhal Conjunctivitis, *Brooklyn Medical Journal*, 1902.

Atropia: Its Uses and Contraindications in Ophthalmic Practice, *Brooklyn Medical Journal*, 1903.

Report of Two Cases of Iritis with Comparative Remarks, *Brooklyn Medical Journal*, 1904.

Re-Attachment in Iridodialysis: A Method Which Does Not Incarcerate the Iris, *Archives of Ophthalmology*, 1909.

Replacement of Incarceration of the Iris by Traction from Within the Anterior Chamber, *Archives of Ophthalmology*, 1909.

Surgical Treatment of Wounds of Cornea with Prolapsed Iris, *Archives of Ophthalmology*, 1926.

Schaaaf Forceps for Removal of Foreign Bodies, *Archives of Ophthalmology*, 1931.

Surgical Entity of Muscle Recession, *Archives of Ophthalmology*, 1931.

Some Essentials and Securities Which Stabilize Operations on Ocular Muscles, *Archives of Ophthalmology*, 1932.

Vascularization of Anterior Segment of Eye: Bearing of These Studies on Some Operative Procedures, Including Possible Supplementary Procedure for Glaucoma; Preliminary Report, *Archives of Ophthalmology*, 1933.

Use of Thyroxin in Ophthalmology: Its Application as a Local Agent and Its Action as Metabolic Alterative, *Archives of Ophthalmology*, 1934.

Ligated Suture, *Archives of Ophthalmology*, 1934.

Some Observations as to Indications for Advancement and Kindred Operations, *Archives of Ophthalmology*, 1935.

Brooklyn Eye and Ear Hospital, 1868-1935, *Archives of Ophthalmology*, 1935.

Operative Treatment of Detached Retina: Principles Observed by Six Individual Operators, *Archives of Ophthalmology*, 1936.

Recession Operation (Reply to Dr. O'Connor), *American Journal of Ophthalmology*, 1936.

Sub-Conjunctival Section of the Ductules of the Lacrimal Gland as a Cure for Epiphora, *Archives of Ophthalmology*, 1937.

Surgical Management of Ptosis with Special Reference to Use of Superior Rectus Muscle, *Archives of Ophthalmology*, 1937.

Technique of Scleral Fixation of Extra-Ocular Muscles, *American Journal of Surgery*, 1938.

Entity of Muscle Recession: Short Résumé of Its Technique and Principles with New Supplementary Notes and Illustrations, *Archives of Ophthalmology*, 1939.

In 1922 he published his paper on "The Correction of Squint by Muscle Recession with Scleral Suturing." This operation is now familiarly known as the Jameson recession operation.

He was regular in attendance at the meetings of his local and national societies and usually contributed to the discussions.

He was tall and slim, soft spoken and kindly in manner, with a genial and cordial personality which endeared him to his friends and associates. The sincerity and staunchness of his character reflected his firm convictions; from these he was not easily swayed. A senior member of his profession, he was most considerate of the younger practitioners, giving to many a helping hand.

Dr. Jameson was an extensive traveler, spending many summers abroad, especially in the British Isles. On such trips he often visited the foreign hospitals and clinics.

A great lover of nature, he sought the wide open spaces and enjoyed long hikes through the country. With a keen sense of humor, he delighted to tell his Scotch stories, and he told them well.

In 1894 he married Miss Sarah Hanmer and had one daughter, Miss Jeanette Jameson, both of whom survive him.

He was a member of the old Hamilton Club of Brooklyn and attended the Fifth Avenue Presbyterian Church in New York.

Dr. Jameson will be remembered by his many friends, patients and associates as a skilled and conscientious physician and as a high type of gentleman with lofty principles and high ideals. JOHN H. OHLY.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

STAPHYLOCOCCUS TOXIN. J. H. ALLEN and A. E. BRALEY, *Am. J. Ophth.* 22: 11 (Jan.) 1939.

After a study of the effects of staphylococcus toxin on rabbits, Allen and Braley reach these conclusions:

"1. The pathogenic action of ocular strains of staphylotoxin is similar to that of general strains, with perhaps the exception of the action upon the gastrointestinal tract.

"2. The hemolytic unit apparently is satisfactory for estimating the potency of ocular staphylotoxin.

"3. Serologic studies should be made upon all rabbits used for experimental study with staphylococci and their toxins."

W. S. REESE.

Biochemistry

TESTS IN CHEMICAL AND PHYSICOCHEMICAL TOPOGRAPHY OF THE LENS. E. TRON, *Vestnik oftāl.* 14: 59, 1939.

This article is extensive and lends itself to abstracting with difficulty. Tron devoted this part of the work (the second part will follow) to the study of the physicochemical topography of the lens, by comparing its anterior and posterior cortical layers and their reaction with similar external factors. "Salt" cataract obtained by immersion of the lens of the bull's eye in hypertonic Ringer-Lock solution served as an object for the study. The difference in the usual technic was that a portion of the sclera and ciliary body was left attached to the lens; a glass curved rod was put through this portion, so that the lens was hanging freely in the solution, and both its surfaces could be seen and examined with the slit lamp.

Different changes were observed on the two surfaces of the lens both in the early and in the late stage. In the early stage of cataract development (caused chiefly by the loss of water by the lens) a large number of large vacuoles were found on the anterior surface of the lens, while on the posterior surface only a few small vacuoles formed, which would soon disappear. The later stage, characterized by the formation of dense chalklike opacities of the superficial layers of the lens with their consequent degeneration, different intensity and different time of development of the process on both surfaces, was observed.

In salt cataract due to immersion of the lens in a copper sulfate solution and mainly to precipitation of the lens proteins, there were also observed different changes on the two surfaces, both in the time of formation of the opacities and in their morphologic structure.

The tests with partial removal of the lens capsule demonstrated first that in the absence of the capsule the opacities varied on the anterior

and posterior surfaces, and secondly, that the opacities varied on the same surface according to the presence or absence of the capsule.

The work is to be continued.

O. SITCHEVSKA.

Comparative Ophthalmology

THE BINOCULAR VISUAL SPACE OF DOMESTIC ANIMALS. A. PISA, Arch. f. Ophth. 140: 1 (Feb.) 1939.

In 1826 Johannes Müller, by rather crude experiments, established the fact that animals with laterally directed eyes have a binocular visual space of considerable size, which fact accounted for the obviously good depth perception of some of those animals, especially birds. In 1902 Tschermak-Seysenegg made measurements of the horizontal expanse of this binocular visual space of various domestic animals. The whole head of the freshly killed animal was frozen, the orbital roofs were removed by careful dissection and the outlines of the binocular field were determined by watching through the sclera the retinal images of a point of light located in front of the animal's head. Perimetric study was thus carried out by direct objective observation of the retinal images. The rabbit, for instance, was found to have a binocular visual space measuring about 34 degrees in its horizontal diameter. Tschermak-Seysenegg concluded that in the visual space of most vertebrates, including animals with total decussation of the optic nerve fibers in the chiasm, there is at least a small area within which sensory cooperation of the two retinas is possible. Thus Tschermak-Seysenegg proved the incorrectness of the concept of a direct proportionality between the number of uncrossed fibers in the chiasm and the extent of the binocular visual space.

Pisa has continued the work of Tschermak-Seysenegg (under the latter's supervision) and has concerned himself chiefly with the question to what extent and in which direction the visual axes of freshly killed domestic animals deviate from their in vivo position. Thereby he arrives, with complicated methods, at figures which probably apply to the practical binocular visual space.

P. C. KRONFELD.

Cornea and Sclera

SUPERFICIAL MARGINAL KERATITIS. O. H. ELLIS, Am. J. Ophth. 22: 161 (Feb.) 1939.

Ellis briefly discusses marginal ulcers of the cornea and superficial marginal keratitis in particular. He reports 2 cases and gives the following summary:

"The first case of superficial marginal keratitis herein described presented severe bilateral involvement, which greatly interfered with vision and caused intense pain, necessitating the enucleation of both eyes. Opportunity was thus offered for describing the anatomical findings in this condition. This is the first time that the anatomical findings of superficial marginal keratitis have been published. A second case with the clinical findings of superficial marginal keratitis is reported."

W. S. REESE.

TREATMENT OF KERATITIS WITH HYPOPYON BY THERMOPUNCTURE OF THE CORNEA. G. PACALIN, Bull. Soc. d'opht. de Paris 50:42 (Jan.) 1939.

Pacalin claims to have been the first to replace the Saemisch section by puncture of the cornea with galvanocautery (*Arch. d'opht.* 48:498 [July] 1931). Puncture relieves the increased pressure in the aqueous, helps in nutrition of the cornea and allows for additional immune substance to be accumulated in the secondary aqueous. The same type of cautery is used as that which the author recommends for retinal detachment. Two case histories are given to show the efficacy of the procedure. The relief of the pain is immediate. If there is any reason to believe that there is infection in the lacrimal tract, the puncta are sealed by galvanocautery. Neither the use of antiseptics nor further surgical procedures are necessary. L. L. MAYER.

MEGALOCORNEA IN CONNECTION WITH OTHER PHYSICAL IRREGULARITIES IN MEMBERS OF THE SAME FAMILY: REPORT OF CASES. R. G. POSTHUMUS, Klin. Monatsbl. f. Augenh. 102:1 (Jan.) 1939.

Posthumus reports 3 cases of megalocornea in a family two previous generations of which had been free from this disease. He found diabetes, labyrinthine deafness and nephritis in several members of this family; the hair was poorly pigmented, especially in the men. Extraction of a nuclear cataract in 1 adult is described, vision of 8/10 having been obtained. In none of the patients with megalocornea was the globe enlarged; atrophy of the vitreous and degeneration of the pigment of the iris occurred. Typical receding of the forehead was noted in some of these patients, but this symptom cannot be regarded with certainty as hereditary. Slight mental debility of these patients cannot be evaluated reliably from a psychic or physical standpoint.

K. L. STOLL.

ULCUS RODENS MOOREN. E. DALSGAARD-NIELSEN, Acta ophth. 17:28, 1939.

This article contains clinical reports of 2 cases of Mooren's ulcer. One eye was enucleated, and the histologic changes are described. These consisted of thinning of the affected portion of the cornea, proliferation of epithelium, destruction of Bowman's membrane and preservation of Descemet's membrane, newly formed vessels and islands of migratory cells. These pathologic changes occurred in portions of the cornea which macroscopically seemed healthy.

O. P. PERKINS.

Experimental Pathology

HISTOLOGICAL STUDY OF THE EYES OF RABBITS CONGENITALLY INFECTED WITH SYPHILIS. H. SHIGA, Am. J. Ophth. 22:119 (Feb.) 1939.

On the basis of an experimental study of the eyes of rabbits congenitally infected with syphilis, Shiga makes the following conclusions:

"One hundred forty-one eyes of 71 rabbits with congenital syphilis, ranging in age from very young fetuses to 93 days, were examined.

Thirty-four eyes of 17 normal rabbits of corresponding ages were taken as controls. The conclusions are as follows:

"1. Congenitally syphilitic rabbits are very liable to be born prematurely by abortion. Even if born at full term, they often die soon after birth. Their general development is below normal, and the development of ocular tissue is deficient as compared with that of normal rabbits.

"2. Pathological changes noted are as follows: (a) In the premature fetus and dead fetus born at full term—edema in the subcutaneous tissue of both lids; hyperemia and hemorrhage in the subcutaneous tissue of both lids, extrinsic ocular muscles, in the neighborhood of the lacrimal gland, conjunctiva, subconjunctival tissue, and the cornea.

"(b) In the young which died shortly after birth—infiltration of round cells in the limbus, the sclerotic, the iris, the ciliary body, and extrinsic ocular muscles.

"(c) In two young (aged 2 months) with corneal ulcer—in one of these I found spirochaetae in the cornea. Also prominent cell infiltration (pseudo-eosinophilic leucocytes) could be seen, especially in the anterior layer of the substantia propria, accompanied by a wide area of epithelial defect. The limbus (remote from the ulcer) showed such syphilitic changes as perivascular cell infiltration, vascularization directly under the corneal epithelium near the limbus, and also in the inner layer of the limbus, with irregular curling of fibrous tissue, thickening of capillary vessel walls, and other changes.

"Epithelial defect with subepithelial change is suggestive of a few cases of keratitis of the human fetus (congenital syphilis cases, or those supposed to be such) reported by H. v. Hippel, Reis, Cattaneo, and Seefelder, and my case is identified as a special type of interstitial keratitis, occurring in congenital syphilis of the rabbit, somewhat different from that in acquired syphilis.

"3. Spirochaetae pallidae were found in the cornea and the sclerotic, sometimes accompanied by pathologic changes, sometimes not."

W. S. REESE.

THE METABOLISM OF THE RETINA AFTER INTERRUPTION OF THE
RETINAL CIRCULATION. A. SANTONI, *Ann. di ottal. e clin. ocul.*
67: 299 (April) 1939.

'The author placed a ligature about the entire orbital contents of rabbits and rats, controlling the changes in the fundus with the ophthalmoscope to be sure that obliteration of the retinal circulation was complete. The ligature was left in place for fifteen to ninety minutes, and the eyes were removed after ten to twelve days and the retina placed in the Warburg respirator, the retina of the fellow eye being used as the control. The oxidative power of the retina showed an increase after interruption of the circulation for fifteen to thirty minutes, while after the ligature was retained for sixty to ninety minutes a decrease in oxidation was noted, averaging 25 per cent of the normal. Santoni believes that the degenerative changes resulting from shorter interruptions of

the circulation free autolytic ferments and fatty acids, which increase oxidation, while after degeneration has reached a certain degree this increased activity no longer occurs and oxidation drops below normal.

S. R. GIFFORD.

General Diseases

BILATERAL RETROBULBAR NEURITIS AS A BEGINNING SIGN OF PERNICIOUS ANEMIA. L. PAUFIQUE, *Bull. Soc. d'opht. de Paris* 50: 144 (Feb.) 1939.

It is not unusual to find retrobulbar neuritis in the advanced stage of pernicious anemia, but visual difficulties are unusual in the early stages. A man of 45 complained of gradual loss of vision for the past thirty days. Visual acuity was 3/10 in the right eye and 3/10 in the left eye. The refraction and pupils were normal, as were the peripheral fields. However, there were bilateral central scotomas for red and green. There was a subicteric tinge. The teeth were badly infected. The patient drank 2 liters of wine a day and was an excessive smoker. A diagnosis of toxic amblyopia was made. In spite of care of the teeth and limitation of smoking and drinking, the condition became rapidly worse. Because of the pallor of the skin and mucous membranes, a complete examination of the blood was ordered. There were 2,000,000 red cells and a hemoglobin content of 42 per cent with an index of 1. There were marked anisocytosis and poikilocytosis. Under treatment, recovery was prompt. In one month the visual acuity had returned to 0.8, and the central scotomas had disappeared.

L. L. MAYER.

Hygiene, Sociology, Education and History

GLASSES FOR PREVENTION OF GLARE IN NIGHT DRIVING. H. VIALLEFONT, *Bull. Soc. d'opht. de Paris* 50: 97 (Feb.) 1939.

Grave accidents may occur because of the glare of the lights from an oncoming automobile. An ideal protection consists of a lens which utilizes all vision in darkness but shuts out glare when it appears. This may readily be arranged by gluing a small segment of neutral glass at the upper right corner of each lens. This segment interferes in no way with direct vision, and only when the head is tilted will the protective glass come into usage. A photograph of the glasses is shown.

L. L. MAYER.

Methods of Examination

A PERFECTION SCOTOMETER. F. A. JULER, *Brit. J. Ophth.* 23: 239 (April) 1939.

The apparatus described by Juler aims at the same results as the Bishop Harman instrument, the test object being a spot of light on a gray surface. The advantage claimed for the instrument is that it is less complicated than the one devised by Harman.

The article is illustrated.

W. ZENTMAYER.

UTILITY OF ANGIOSCOTOMETRIC EXAMINATION IN THE DIAGNOSIS OF
EDEMA OF THE PAPILLA. A. MAGITOT and A. DUBOIS-POULSEN,
Bull. Soc. d'opht. de Paris 50: 32 (Jan.) 1939.

Angioscotometric examination has been used with much success in the study of the vascular tree of the retina. With a 0.5 mm. test object, deviation in the extent of Mariotte's blindspot are well delineated. In this article the authors report on a study of pathologic blindspots of Mariotte. Factors influencing the size were found to be voluntary apnea, cervical constriction, previous use of the electric light of the ophthalmoscope and pressure on the globe.

In cases of hyperopia with pseudoneuritis, the blindspots were found to be normal by angioscotometric examination. If any enlargement occurred in such cases, search for the cause of hyperemia of the disk was sought. Frequently, as demonstrated by Evans, hyperemia of the disk with enlargement on angioscotometric examination has been found to be due to involvement of a sinus.

In endocrine disorders and even at menstruation, edema of the nerve head occurs with enlargement of the blindspot. In stasis and papillitis, minute changes in the outline of the disk are readily found with angioscotometric examination, and it is relatively simple to watch the progress of an increased intracranial pressure by this method.

As a matter of fact, the authors believe that this form of examination is the only one which distinguishes between stasis and papillitis in many cases. One is better able to distinguish the absolute central blind area and the peripheral amblyopic zone by his method. The authors cannot decide whether Evans' hypothesis is justified, in which he attributes the amblyopic zone to the presence of a large number of small vessels which arch over the disk.

L. L. MAYER.

Neurology

FACIAL HEMIATROPHY. F. B. WALSH, Am. J. Ophth. 22: 1 (Jan.) 1939.

Walsh reports 2 cases of facial hemiatrophy illustrating the two types, congenital, or nonprogressive, and the more frequent progressive type. He reviews the theories explaining this condition and discusses its relation and resemblance to scleroderma. The second case reported was unique in that heart block was present. Walsh enumerates the ocular signs that occur, though these are not constant.

W. S. REESE.

OCULAR SYMPTOMS IN SCHIZOPHRENIC PATIENTS TREATED WITH
METRAZOL (CARDIAZOL) AND WITH INSULIN SHOCK THERAPY.
V. CAVKA, Arch. f. Psychiat. 109: 721 (April) 1939.

Ten schizophrenic patients who were treated with metrazol (cardiazol) and 20 who received insulin shock therapy were studied for ocular changes before, during and after the convulsion. Of the first group, no abnormalities of the fundi were evident before treatment.

Six were emmetropic, 4 were hyperopic and 1 was myopic with conus. Cavka demonstrates his observations on this group in a table. The outstanding characteristic was blepharospasm with conjugate deviation to the right and upward during the reaction. The pupils were widely dilated. The retinal arteries were reduced to one-fourth their caliber, and the veins were dilated, so that the relation before the reaction was 1:2.5 instead of 1:1.5.

In 2 patients there was a blurring of the outlines of the disks, which disappeared several days after the reaction. No hemorrhages were noted. Cavka feels that such symptoms are comparable to the usual attack and changes in the fundus in idiopathic epilepsy.

The group given insulin therapy showed similar changes. The intra-ocular tension of 13 persons in this group was raised.

L. L. MAYER.

PERMANENT LESION OF THE VISUAL TRACT DUE TO FLYING AT HIGH ALTITUDES: REPORT OF A CASE. W. LÖHLEIN, *Klin. Monatsbl. f. Augenh.* 101: 818 (Dec.) 1938.

The patient, aged 34, who had been an aviator for several years, had escaped death in an aerial accident in 1937 in which his companion died. A few weeks later, ascending to an altitude of from 5,000 to 6,000 meters, he suddenly experienced severe headache, vertigo and obscuration of the field of vision. He was barely able to land his aeroplane. His companion showed no symptoms except severe epistaxis, while the patient himself, after landing, presented vertigo, nystagmus, Romberg's symptom, right hemianesthesia and right homonymous hemianopia due to total scotomas in the central zone of each eye. Few neurologic symptoms were noted. The ocular symptoms were unchanged after a period of rest of three months. Vision in each eye remained normal. Löhlein interprets the symptoms as the result of a lesion of the left cerebral hemisphere, possibly toward the left inner capsule. The sudden onset, the absence of an increase of the symptoms, the fairly negative roentgenographic findings and the negative result of the test of the spinal fluid are indicative, in the author's mind, of a vascular disturbance in the left cerebrum, which may have been a vascular spasm or a hemorrhage. Lack of oxygen and fast ascendance may be the source of the symptoms in both men. The graver disturbance in the patient described may be attributed to individual susceptibility or perhaps to some kind of a mechanical lesion of the blood vessels as a result of the accident suffered four months previously.

K. L. STOLL.

EXOPHTHALMOS COINCIDENT WITH INTRACRANIAL TUMORS. H. SKYDSGAARD, *Acta ophth.* 16: 474, 1938.

The author limits his remarks to tumors arising from the cerebrum, meninges and cerebral vessels. Exophthalmos was found in 14 of a series of 352 patients with such tumors, seen at the Rigshospital. The exophthalmos was unilateral in 9.

The tumors are tabulated as follows:

Meningioma of the pterygoid bone.....	5	{(unilateral 4) (bilateral 1)}
Parasagittal meningioma	1	(bilateral)
Convexity meningioma	1	(unilateral)
Meningioma of the olfactory nerve.....	1	(bilateral)
Neurinoma of the acoustic nerve.....	1	(unilateral)
Tumor of the hypophysis.....	3	(unilateral)
Aneurysm of the carotid artery.....	1	(bilateral)
Glioma of the temporal lobe.....	1	(bilateral)

Similar sets of figures from the published series of other authors are given.

The author remarks that while papilledema and exophthalmos may occur together, they by no means necessarily do so; nor does exophthalmos seem to have any definite relation to increased intracranial pressure.

O. P. PERKINS.

CHANGES IN THE FUNDUS OCULI AND PERSISTING INJURIES TO THE EYE IN MIGRAINE. H. GRONVALL, *Acta ophth.* 16: 602, 1938.

An 18 year old girl who had suffered from typical attacks of migraine for six years suddenly became blind in the right eye after a prodromal period of giddiness and scintillations. Ten minutes later the lower portion of the visual field was restored, a defect above remaining. Ophthalmoscopic examination twenty-four hours later revealed an area of retinal edema and obliteration of the inferior temporal branch of the central retinal artery. The edema gradually subsided, but the arterial change persisted during the two months that the patient was observed.

No source of an embolus could be found, and the case was regarded as one of arterial spasm associated with migraine.

O. P. PERKINS.

Ocular Muscles

CONTRIBUTION TO THE STUDY OF ACQUIRED SPASMS OF THE LEVATOR MUSCLE OF THE UPPER LID. G. E. JAYLE, *Ann. d'ocul.* 176:1 (Jan.) 1939.

In spite of numerous researches on acquired spasms of the levator muscle of the upper lid, the particular characters of this condition as well as the symptoms are not well understood. The question of the pathogenesis also remains in dispute.

Its position among other retractions of the upper lid is not precise, some investigators placing it with the palpable retractions. Certain authors claim that the palpable retractions of the striated type and the retractions of the sympathetic type are attributable to identical lesions.

The main division of the article deals with binocular spasms of the levator muscles of the upper lid, which material is subdivided into that dealing with permanent and intermittent retractions; the symptoms associated with former, such as oculomotor disturbances, Parinaud's syndrome, peripheral paralysis, pupillary changes, exophthalmos, vestibular disturbances, oculocephalic symptoms and general disturbances, and pathogenesis.

The intermittent retractions are discussed under synkinetic spasms, effort spasms and fixation spasms.

The article is to be continued.

S. H. MCKEE.

Pharmacology

PONTOCAINE, THE CAUSE OF PROFESSIONAL ECZEMA AMONG OCULISTS.
N. SHIMKIN, *Ann. d'ocul.* 176: 198 (March) 1939.

Pontocaine is an anesthetic produced to replace cocaine. It is a derivative of procaine, plus a radical "butyl," the ethyl group being here replaced by the methyl group.

The author reports a case in which palpable eczema developed after the instillation of 1 to 2 drops of a 0.5 to 1 per cent solution of pontocaine hydrochloride. The condition developed within two to three hours and was manifest by a burning sensation in the eyes with redness and edema of the skin of the lids. After six to eight hours a typical eczema of the lids appeared. This condition lasts from eight to fourteen days but disappears completely with treatment by cold compresses. A number of cases of eczema of the fingers, particularly of the nails, have been observed in oculists after the use of pontocaine.

The author himself suffered from eczema of the hands and of the left leg during four years' use of the drug in his clinic. The condition was completely corrected when pontocaine was no longer employed. Other instances are cited similar to the author's own experience.

S. H. MCKEE.

ACTION OF ATROPINE ON THE ENUCLEATED EYE OF THE FROG. J. RIGNIER and A. QUEAUVILLER, *Compt. rend. Soc. de biol.* 130: 1212, 1215 and 1461, 1939.

The enucleated frog's eye kept in mild diffuse light regularly becomes miotic, both in Ringer's solution and in isotonic dextrose solution, reaching its limit by the second hour. After one hour the pupillary area is generally reduced by 30 per cent. The relative action of atropine on the enucleated eye is most marked in the first hour and is greater in Ringer's solution than in isotonic dextrose solution. In the same medium and at the same p_H the mydriatic action of the sulfate is twice that of the citrate or the phenylpropionate salt. Since none of the negative ions (chloride, citrate, sulfate or phenylpropionate) have any topical effect on the pupil, the specific properties of the anion hence do not explain the differences in action of the various salts of atropine.

J. E. LEBENSOHN.

The Pupil

ARGYLL ROBERTSON PUPILS IN ALCOHOLISM. M. HERMAN, *Arch. Neurol. & Psychiat.* 41: 800 (April) 1939.

The author reports 3 cases illustrating the association of an Argyll Robertson pupil with chronic alcoholism. The summary follows:

"In case 1 there was evidence of widespread damage to the central nervous system, as shown by the Korsakoff type of mental change, the peripheral neuritis and the pellagrous lesion. In case 2 the classic pupillary change was found, together with extensive peripheral neuritis in the legs. In case 3 there was a transitory pupillary reaction of the Argyll Robertson type during the course of delirium tremens. Wherever the pathologic lesion that is responsible for an Argyll Robertson pupil

exists in cases of neurosyphilis, it seems that a similar pupillary change may be produced in other conditions. In cases of chronic alcoholism, the Argyll Robertson pupil was found accompanying other neurologic disturbances. It is interesting to note that this manifestation may be transitory, as in case 3. Possibly, with repeated attacks a permanent pupillary change may result."

R. IRVINE.

Physiology

THE CHEMICAL EQUILIBRIUM OF THE INTERSTITIAL FLUIDS AND THE AQUEOUS HUMOUR. J. D. ROBERTSON, *Brit. J. Ophth.* 23:170 (March) 1939.

This article is limited to the discussion of the chemical equilibrium that exists (1) between blood and lymph, (2) between blood and gastric juice and (3) between blood and aqueous humor.

The following summary is given by the author: "I have already stated, glucose, urea, and creatinine are freely diffusible and entirely filtrable both in vivo and vitro so that if the membrane in the eye and the choroid plexus allowed simple dialysis to occur, these substances should occur in concentrations equal to that in the plasma; but they do not. It would appear in fact that glucose, urea, and creatinine, diffuse through the stomach wall with no less difficulty than they diffuse into the aqueous humor. In both the stomach and the eye, there is a marked deficiency in the urea, sugar, and creatinine concentrations when these are compared with the concentration of these substances in the blood. In the case of creatinine, all the experiments showed that this substance diffused into the stomach much more readily than into the aqueous humor. It has been stated that it is a second membrane re-inforcing the capillaries of the ciliary processes which is responsible for the low protein content of the aqueous humor, and the impaired filtration of freely diffusible substances such as urea, glucose, creatinine, and uric acid. This so-called reinforced membrane behaves not unlike the secreting membrane present in the stomach, for it causes a similar concentration gradient for diffusible substances.

"When all these facts I have mentioned are considered, it becomes clear that we cannot consider the aqueous humor a simple, protein-free ultrafiltrate or dialysate of blood plasma, but that it is a highly specialised fluid manufactured for a specific purpose. As we found the simple laws governing the formation and absorption of tissue fluid inapplicable to the production of the aqueous humor, so we find the chemical analysis too complex to be explained by the simple laws governing the chemical equilibrium of dialysates." W. ZENTMAYER.

Retina and Optic Nerve

SOLITARY RETINOCHOROIDITIS WITH REFERENCE TO RETINOCHOROIDITIS JUXTAPAPILLARIS-JENSEN. C. HEATH, *Brit. J. Ophth.* 23:289 (May) 1939.

Heath prefers the term solitary retinochoroiditis for the disease to which Jensen's name is usually given. Attention is called to the fact that six years prior to Jensen's paper H. Friedenwald gave a far more

comprehensive description of similar cases and mentioned that the condition had already been recognized by Griffith. It is proper to state, however, that Jensen restricted the application of the term retino-choroiditis juxtapapillaris to those cases in which the lesion is in contact with the disk and produces a sector-like defect in the field of vision. Heath describes a typical case of disease as follows:

An apparently healthy young adult complains that the sight of one eye has been blurred for two or three days. The vision in that eye is found to be reduced to about 6/12. External examination shows nothing amiss. Examination of the fundus shows a vitreous haze which obscures the finer details, but close to the disk is seen a patch of shining, cream-colored exudate shading off gradually into the normal retina on all sides. A week or more later the vision in that eye has deteriorated to about 6/36. Fine keratic precipitates are present, and the vitreous haze is thicker than before. The exudate and the rest of the fundus appear unchanged. The condition remains the same for one or two weeks and then steadily improves. In three months the media are clear, and vision is restored to normal. The fundus shows a pigmented scar corresponding to the site of the exudate, but it is otherwise normal. The field of vision shows an absolute sector defect extending from the blind spot to the periphery.

Twenty-four cases were investigated and the findings compared with those recorded in the literature. An attempt has been made to define criteria for diagnosis. Points of particular interest which emerge are:

"1. The apparent high incidence of the disease in childhood.

"2. The frequent presence of veils in the vitreous.

"3. One case in which the development of anomalous retinal vessels was preceded by retinal ischaemia.

"4. The defects in the field of vision may be wholly or partly relative and may diminish with healing.

"5. One case in which a defect in the visual field was relative throughout after the first attack but became absolute after the second.

"6. One case in which two separate sector-defects in the visual field were produced by two apparently confluent scars.

"7. Defects originating in the nasal visual field appear to be incompatible with the conventional conception of the distribution of the nerve-fibres of the retina."

Heath gives a detailed discussion of the subjective and objective symptoms of the disease as found in his own case reports and those in the literature.

The article is illustrated.

W. ZENTMAYER.

ENDOCRINOLOGIC REPORTS ON RETINITIS PIGMENTOSA. E. BIRÓ, *Ann. d'ocul.* 176: 293 (April) 1939.

It is only within the last ten years that pigmentary degeneration of the retina has been investigated from an endocrinologic point of view. Up to this date most research has been along anatomicopathologic lines, a satisfactory explanation of the cause and origin of the pigmented retina being sought in histologic study. Investigators of the last thirty

years have searched for, and are still seeking to find, evidence to show whether the displaced pigment is of choroidal or of retinal origin and whether the first changes in the disease are in the choriocapillary layer of the choroid or in the neuroepithelial layer of the retina.

The author's ideas on the causation of retinitis pigmentosa have been influenced by the finding of a relation between the endocrine glands and pigmentary retinitis. Experimental research is being continued with extracts of ovary and testis and with liver extracts. These experiments and their results are to be published later.

S. H. McKEE.

Trachoma

BEE VENOM IN THE TREATMENT OF TRACHOMA. A. LAGOMARSINO, *Ann. di ottal. e clin. ocul.* 67: 143 (Feb.) 1939.

The preparation of bee venom, as described by Brecher, and the literature on its use in trachoma are reviewed. Twenty patients with trachoma in various stages were given subconjunctival injections of the venom mixed just before injection with a local anesthetic. Half an ampule was injected in the upper fold and the other half into the tarsal conjunctiva and tarsus. In cases in which there were pannus and corneal lesions, half an ampule was injected under the bulbar conjunctiva near the limbus. Six injections were usually given at intervals, depending on the duration of the reactions. Treatments were not painful and were well tolerated. The effects were favorable, especially on the corneal lesions, which cleared rapidly. The vessels in some cases disappeared, and corneas that were affected by thin opacities became much clearer. Corneal ulcers healed under treatment, with minimal scarring.

S. R. GIFFORD.

Society Transactions

EDITED BY W. L. BENEDICT

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

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Forty-Fourth Annual Meeting, Oct. 8-13, 1939

Essential Hypertension from the Standpoint of Otolaryngology.¹

DR. A. C. FURSTENBERG, DR. J. M. MAXWELL (by invitation) and
DR. G. H. ALEXANDER (by invitation), Ann Arbor Mich.

While the cause of essential hypertension is not yet clearly understood, a distinct hereditary tendency is recognized, as shown by the numerous reports of cardiovascular symptoms or accidents which appear in the family histories.

From a practical standpoint, it is of utmost importance that the otolaryngologist recognize the occurrence of varying degrees of hypertension and the fact that high blood pressure is not a constant finding in the early stages of the disease. Only repeated examinations may reveal the occurrence and extent of the disease, as the patient frequently consults the otolaryngologist on account of headache. This symptom dominated the clinical picture in 50 per cent of 156 cases of essential hypertension that were extensively studied at the University of Michigan Hospital. Many of the patients reported a previous diagnosis of sinus disease, but only 12 per cent had pain that was frontal in character and of the pressure type that might be confused with the pain of sinusitis. The headache was frequently nocturnal, dull and boring, reaching its maximum intensity in the early morning and vanishing completely in the afternoon. Invariably the patient stated that the headaches were aggravated by emotional excitement, nervousness and fatigue.

Another common symptom of hypertension and, next to headache, the most annoying disturbance of the disease is vertigo. It was presented by 30 per cent of the patients. It is usually described as a feeling of unsteadiness or a "swimming of the head" and is often initiated by a sudden change of position or a quick motion of the head; it is ordinarily relieved by a short period of rest.

It was found that nearly all of the patients presented emotional disturbances or periods of irritability, anxiety and apprehension. One of the

1. This paper and the following two on essential hypertension were presented as a symposium on this subject at a joint session of the Section on Ophthalmology and the Section on Otolaryngology.

most annoying and occasionally terrifying experiences is epistaxis. This symptom occurred in 17 per cent of the cases. The bleeding comes from the septal branch of the sphenopalatine artery, where it anastomoses with a branch of the descending palatine artery on the anterior portion of the quadrangular cartilage.

It is an astonishing fact that patients with hypertension tolerate surgical intervention admirably well. Care should be taken to avoid unnecessary traumatism of tissues and careful attention should be given to ligation of blood vessels of large enough caliber to favor hemorrhage. If a general anesthetic is required, ether, perhaps, administered by the open or intratracheal technic, is the safest. Danger from the anesthetic is minimized if the blood pressure can be maintained at a nearly constant level during operation. To do this it occasionally becomes necessary to administer fluids intravenously and some vasopressor agent to increase the pressure, which has a tendency to fall while the operation is in progress. The most useful drug of this character is neo-synephrin hydrochloride, administered in doses of 0.5 cc. (5 mg.) either intramuscularly or intravenously.

Audiometric determinations were made in this series of 156 cases of hypertension, in 154 of which a diagnosis of essential hypertension was made while in 2 the diagnosis was malignant hypertension. Significant loss of hearing was demonstrated in 148 cases, or 96.1 per cent. The deafness, which was bilateral in nearly all cases, manifested a striking parallelism in the amount of loss of hearing and the tone frequencies involved in both ears. There seemed to be no parallelism between the duration of the hypertension and the degree of deafness.

An opportunity for further study of deafness in cases of hypertension was afforded in 37 cases of this series in which the patients submitted to splanchnicectomy for reduction of the blood pressure. Audiograms were made three months after the operation and compared with the preoperative charts. In most cases there was a striking decrease in the hypertension after the operation, and this was associated with some improvement in the hearing. However, it was not possible to demonstrate any correlation between the degree of improvement in the auditory function and the amount of reduction in the blood pressure after section of the splanchnic nerves.

Essential Hypertension from the Standpoint of Ophthalmology.

DR. HENRY P. WAGENER, Rochester, Minn.

At this time the retinal pictures typical of chronic or rapidly progressive hypertensive disease have not been reproduced in characteristic form or course in experimental animals. In spite of the inadequacy of present experimental evidence, advances are being made in the understanding of the mechanism involved in the production of the retinal lesions and of their relation to the systemic, pathologic and pathologic-physiologic processes of the hypertensive person.

The conception that a toxic substance circulating in the blood is responsible for the lesions in the retina commonly designated as "retinitis" and that the kidneys are the sources of this toxic substance has been most widely accepted. It has been impossible thus far to demonstrate the existence of such a toxic substance.

Clinically, it seems obvious that the most severe forms of retinal vascular disease and of retinitis can develop in the course of hypertensive disease in the absence of any marked or even definite evidence of renal impairment. When severe renal insufficiency occurs in association with retinitis in patients who have primary hypertension, it seems probable that the damage to the kidneys is not the essential feature of the disease but that it is only a part of widespread injury to the vascular system.

In cases of "albuminuric retinitis" fairly widespread sclerosis of the arterioles has been demonstrated to be present in the retina at necropsy, but it seems unlikely, both from the clinical and from the histologic standpoint, that organic damage to the retinal vessels can be held responsible for the development of the forms of retinitis characteristic of hypertensive disease. It seems most logical at present to assume that retinitis develops in some manner as a form of decompensation of the retinal circulation and not from organic lesions in the walls of the vessels.

In general, the changes observed ophthalmoscopically to occur in the retinal arterioles in association with hypertensive disease may be grouped under five main subdivisions: (1) generalized smooth narrowing of the lumen, (2) changes in the color, reflex stripe and visibility of the walls, (3) arteriovenous compression, (4) irregular localized constriction of the lumen and (5) periarterial sheathing. In many cases various combinations of these changes are seen. Each of them can occur alone, however, and the significance of each should be considered separately. If the color changes consequent on changes in the blood itself are excluded, it seems probable that changes in the color of the wall of the vessel are due to an actual structural lesion in the wall, which is most often hypertrophy of the media. Irregularly placed, localized narrowings of the lumen of the arterioles have been demonstrated definitely in many instances to be of a transitory spastic, rather than of a permanent organic or structural, nature. The ultimate proof of the spastic nature of any localized narrowing rests, of course, on its complete disappearance. In rapidly advancing or active hypertensive disease, such as that seen in association with acute toxemias of pregnancy, shifting in the situation and degree of these localized regions of narrowing can be observed from day to day. As the hypertension becomes more fixed, the spastic narrowings may become more fixed in situation also.

In most cases, however, the size of the arterioles in the retina is most probably an expression of vasomotor tone, and reduction in their size below that of the average normal indicates an increase in their tonicity. That this is true is suggested by the fact that the diameter of the arterioles corresponds in a general way to the height of the diastolic blood pressure readings. Repeated observations in individual cases demonstrate that the size of the arterioles is not constant but may fluctuate with variations in the blood pressure. Thus, the caliber of the retinal arterioles may be seen to increase after splanchnic sympathectomy for the relief of hypertension.

The picture of localized groups of punctate exudates and hemorrhages, described under the name of "retinitis of arteriosclerosis," represents the end phase of venous obstruction or thrombosis, usually a feature of the cases of milder hypertension. It may also be seen in the terminal phases of "angiospastic retinitis," a term used to designate

types that have been known as "albuminuric." When acute angiospastic retinitis occurs in the presence of chronic sclerosis of the arterioles, the prognosis for the life of the patient is always more serious. Should a person have acute angiospastic retinitis with chronic sclerosis of the retinal arterioles, it means always that he has diffuse arteriosclerosis, no matter whether his primary disease is "essential hypertension" or "chronic nephritis." The retinitis can be regarded only as a manifestation of decompensation of the retinal circulation.

Essential Hypertension from the Standpoint of the Internist. DR. ROY W. SCOTT, Cleveland.

In primary or essential hypertension there is a persistent elevation in both the systolic and the diastolic blood pressure, unrelated to primary renal disease, endocrine or vasomotor disorders. Goldblatt and his associates have clearly demonstrated the renal origin of arterial hypertension. Present day concept of clinical hypertension as presented in most textbooks on the subject may be summarized as follows: 1. Hypertension admittedly of renal origin is associated with glomerular nephritis, urinary obstruction, polycystic kidney disease and periarteritis nodosa with marked renal involvement and severe renal amyloidosis. 2. Essential hypertension is not generally believed to be of renal origin, because there is no demonstrable renal excretory insufficiency in the majority of cases; most patients with essential hypertension die of heart failure or of a cerebral accident without ever showing renal insufficiency; occasionally a case of essential hypertension is reported in which no anatomic evidence of renal arteriolar disease was exhibited.

Since Goldblatt's demonstration of the role the kidney plays in causing arterial hypertension, several cases of hypertension have been reported in human beings in which the renal artery was narrowed or completely occluded and in which on operative removal of the ischemic kidney the blood pressure returned to normal.

Diabetic Retinitis. DR. JONAS S. FRIEDENWALD, Baltimore (from the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital).

General Hypertension and Peripheral Optic Pathways. PROF. DR. JOSEPH IGRERSHEIMER, Istanbul, Turkey (by invitation).

The pathologic processes in the trunk of the optic nerve and on the chiasma in cases of general hypertension (mostly in cases of malignant hypertension) are more frequent and more variable than one believes generally, and they warrant more attention.

Whether the alterations in the connective tissues, such as lymphocytic infiltrations around the central artery and in the septums or peculiar proliferations of the connective tissue in the axial fasciculus, are intimately connected with general hypertension must be left open to question. I have seen such alterations several times in different degrees, and they demonstrate that changes are taking place behind the globe which cannot be recognized clinically.

It is of more importance that degenerative processes in the optic nerve can also occur latently (without ophthalmoscopic signs). It is

probable that they cause sometimes, according to their situation and extension, disturbances of the visual functions. Furthermore, it is remarkable that retinal lesions of especially long duration by no means produce an ascendant degeneration but, on the contrary, just those forms which show clinically and anatomically few typical retinal spots. Instead of this, the small retinal vessels show high grade arteriolo-sclerosis with obliteration of their lumen and lead in this way to severe disturbances at the inner layer with involvement of the nerve fibers.

Moreover, it can be regarded as certain that processes in the optic nerve and in the chiasm may occur which are not dependent on the retinitis albuminurica. Retinitis and processes of the peripheral optic pathway can occur concurrently, as is evident especially in a long observed case of retinitis associated with bitemporal hemianopic scotoma.

It seems that such independent diseases of the peripheral optic pathways can occur in association with different clinical conditions, such as circulatory disturbances and glaucoma-like cupping, and, moreover, in the form of a rapid decrease of the vision combined with neuritic atrophy. Observations have been made which demonstrate these several possibilities, and in most instances clinical and anatomic studies have been made. It is deplorable that the manner of the development of these diseases is often still undiscovered. It is possible that the changes in the small vessels are of great importance, and some of the statements made can be considered from this point of view, but absolute proof of such a connection has not heretofore been obtained. It is to be hoped that further systematic researches and favorable cases may lead to a more certain comprehension.

Acute Hydrops of the Cornea Complicating Keratoconus. DR. RALPH O. RYCHENER, Memphis, Tenn., and DR. DANIEL B. KIRBY, New York.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

Transplantation of the Cornea by Means of a Mechanically Obtained Segment with a Beveled Edge. DR. MEYER WIENER and DR. BENNETT Y. ALVIS, St. Louis.

Ever since transplantation of cornea has been seriously considered by ophthalmic surgeons, they have sought a mechanical means of obtaining a graft with a beveled edge. We have succeeded in designing a punch by means of which a circular segment of cornea having a beveled edge is punched out, the epithelial surface of which is about 0.6 mm. wider than the endothelial surface. The graft is not injured, as shown by macroscopic and microscopic observation. The punch is constructed so that the female blade is 6.5 mm. wide and 1 mm. thick. The upper surface of its bore measures 4.5 mm. in diameter. The under surface is wider, as the hole is cut on a bevel so as to make the upper cutting edge sharp. The male blade is bullet nosed, its diameter, 2.5 mm. from the tip, being the same as that of the hole in the female blade. When the sharp edge of the female blade, after being introduced into the anterior chamber, engages the under surface of the cornea, the tip of

the male blade presses down, reverses the curvature and stretches the entire anterior corneal surface, driving a wider portion of it into the punch. The graft is held in place by fine silk sutures properly placed and loosely tied. When the cornea of the host is vascular, an erythema dose of roentgen rays is given the following day and is repeated a second or even a third time if the vascularity seems to encroach on the graft.

DISCUSSION

DR. VIRGIL G. CASTEN, Boston: Corneal transplantation was first attempted about one hundred and sixteen years ago. In the early years, lack of both asepsis and anesthesia caused many failures. During the past century anesthesia and the technic of corneal transplantation have both greatly improved. Almost all the total corneal transplantations first done by Wagenmann in 1888, when the entire cornea and some conjunctiva were transplanted, resulted in failure.

Von Hippel in 1888 tried so-called circumscribed lamellar corneal transplantation, but connective tissue formed at the base of the transplant, preventing visual success. He then went back to his earlier method of 1877 of partially penetrating corneal transplantation, which Elschnig later found to be the most dependable method. Von Hippel used a trephine to cut his transplants.

Elschnig modified von Hippel's technic slightly and reported improvement in vision in many cases. Filatov further modified von Hippel's operation, making a puncture and counterpuncture of the cornea, through which a strip of celluloid was passed to prevent injury to the iris and lens when the cornea was trephined.

Thomas in 1930 used a 4 to 4.5 mm. trephine, held at an angle of 30 to 45 degrees, so as to penetrate at one side. The trephine was then withdrawn and the button completed with scissors. In this way the inner surface was made smaller than the outer, giving a beveled edge which prevented the transplant from falling into the anterior chamber.

Castroviejo in 1932 used a double-bladed knife, making a rectangular transplant, beveled on one side with a keratome and on the other three sides with scissors, as he found rectangular transplants easier to bevel than round ones.

All of these operations have been time consuming. Drs. Wiener and Alvis have ingeniously devised a new technic for securing uniform, mechanically obtained, beveled grafts, which can be cut in a second and transferred from donor to host. The average ophthalmic surgeon can cut uniform beveled grafts with this instrument. However, with this technic another 8 mm. corneal incision is necessary, which has not been used in any of the former operations. This should not be a great stumbling block.

In the way of criticism, it would seem to me the instrument could be used more easily if it had a bayonet end, so that the brow would not interfere, and also an infinitely thin sleeve to prevent the graft from falling into the anterior chamber when it is cut. Further, it would seem that the graft would be subjected to great trauma, and consequently there would be a higher percentage of opaque transplants by this method. This, however, remains to be seen, as only one operation has been reported.

The type of suture is excellent. Conjunctival flaps have more or less been given up because of the higher incidence of infections, and a suture of some type is being generally used.

DR. RAMON CASTROVIEJO, New York: Drs. Wiener and Alvis deserve to be congratulated for this ingenious instrument, which it is claimed will permit the average ophthalmologist to obtain mechanically a beveled graft.

In the operation described an incision is made at the limbus, through which one blade of the instrument is introduced into the anterior chamber. It is reasonable to suppose that this extra incision at the limbus will lead to complications, such as incarceration of the iris. This supposition is corroborated by the report of the only case in which this technic was used, and in this instance incarceration of the iris took place. Of course a single case is not sufficient from which to draw conclusions. Many more persons will have to be operated on, possibly several hundred, before definite conclusions can be drawn.

The condition of the anterior chamber must also be considered in performing keratoplasty with the technic of Drs. Wiener and Alvis. If the eye is aphakic, the use of the punch described may lead to luxation of the dissected leukoma into the vitreous cavity.

The simplified suture used by Drs. Wiener and Alvis is just one half of the cross stitches advocated by Tudor Thomas. I have observed, however, in the illustrations showing the different steps of the technic that the suture is placed too far from the edge of the incision. It has been the experience at the Institute of Ophthalmology, Columbia-Presbyterian Medical Center, where about 200 keratoplastic operations have been performed, that the sutures must be placed very near the edge of the incision; otherwise, in cases in which the cornea is extremely thin, especially in cases of keratoconus, the cornea collapses and it is difficult or impossible to maintain the transplant in position.

When the cornea is favorable for keratoplasty, a simplified stitch, such as the one advocated by Drs. Wiener and Alvis, may be sufficient. It is, however, good judgment to consider every case a difficult one, in order to avoid unpleasant surprises during the operation and complications during the postoperative course.

A beveled transplant seems to be obtained consistently and easily by the procedure described by Drs. Wiener and Alvis, and according to their statement the edges are cleanly cut, an observation confirmed by microscopic examination.

The ingenious operation presented here deserves to be thoroughly studied. A comparative study of this and of other methods of operation made on eyes of animals will give new data to report in the future as to the real value of the operation.

DR. B. Y. ALVIS, St. Louis: We realize that Dr. Castroviejo's last point is well taken, indeed. One case is not sufficient on which to draw any conclusion and much more study is necessary both to perfect the technic and to prove its value. This paper, however, was given rather as a preliminary report, because we felt that this contribution was sufficiently valuable in that it simplified the technic of corneal transplantation so much that it would be worth while to let others use it and attempt to perfect the technic along with us.

The simplicity of the technic, it seems to me, will make it possible for keratoplasty to be carried out far away from Dr. Castroviejo and others who have had more extensive experience with this type of operation. The length of time required for the operation is only fifteen or twenty minutes.

Some one inquired as to where the punch is made. It is made by the Storz Instrument Company.

The statement was made that the graft would be difficult to recover from the anterior chamber. Such has not proved to be the case. There were a few instances in experimental work that the graft did slip into the anterior chamber, but there was little difficulty in recovering it because of the large incision in the cornea. In fact, the graft is not often lost, and I do not believe that it is more likely to drop into the anterior chamber than if one used a trephine or the double corneal knife of Castroviejo.

The objection occurred to us that probably the graft would be crushed, but when one considers the cutting operation of this punch one notes that the margin is cut in exactly the same way as by the scissors. A microscopic study of such a section showed no visible evidence of crushing of the corneal tissue.

Relation of Vitamin Deficiency to Tryparsamide Reaction. DR.

WILLIAM M. MUNCY, Providence, R. I.

The most satisfactory treatment of neurosyphilis is thought to be fever therapy followed by the administration of tryparsamide. As the fear of ophthalmic complications seems to be an objection to its use, a thorough study of each case before and during treatment seems most important. This investigation was carried out at the Charles V. Chapin Hospital of Providence, R. I. The findings in the first 40 cases were published in the *Rhode Island Medical Journal* (20:169, 1937). A reaction to this drug occurred in 6 of the cases, or 15 per cent. In 5 of these the patients had normal visual and form fields prior to the reaction, and there was nothing on ophthalmoscopic examination that would indicate any disease of the tissue viewed. On the other hand, among the other 34 patients were some with conditions such as old iridic adhesions, retinochoroiditis and even optic neuritis, who were given the drug without harm. From these findings I concluded that the previous state of the eyes was no indication of susceptibility to this drug. Furthermore, the fact that no objective signs were found on examination of the eyeground in these 6 cases during an attack led one to suspect that I was dealing with toxic amblyopia caused probably by vitamin deficiency. Two of the 6 patients who had adverse reactions were again treated with tryparsamide, after having been built up in health, and have had a number of series of treatments since without any untoward effects.

From the summer of 1937 to date, vitamin B and B complex have been given in a second series of cases, 50 in number, in which tryparsamide was administered. The vitamins were given constantly from the time of the patients' admission to the hospital, during fever therapy, throughout the series of treatments with tryparsamide and whenever the administration of the drug was resumed. A reaction occurred in only 1 of the cases. This was a case of atrophy of the optic nerve, probably due to retrobulbar neuritis; after tryparsamide shock, the

patient was brought back to his previous state by numerous intravenous injections of vitamin B. Since then the patient has undergone seventeen treatments with tryparsamide without any further reaction.

In addition, during this period a number of persons who had already had a reaction from tryparsamide were sent to the hospital for treatment. They had not received vitamins as a preventive treatment, but under active vitamin therapy their vision had been restored to its previous state. Among these there was a patient who had a secondary reaction when the administration of tryparsamide was resumed. This reaction followed the third treatment with tryparsamide in spite of numerous intravenous injections of vitamin B. There was a rapid loss of vision and form field in the left eye and complete blindness in the right eye at the time the patient died, twenty-one days after the last treatment. Autopsy disclosed marked arteriosclerosis throughout the brain, with numerous areas of anemia and soft degeneration. The extensive pathologic process found indicated that the cardiovascular disease had already reduced the life expectancy to a narrow margin at best.

These 2 patients were the only ones having had vitamins as a preventive treatment who reacted adversely to tryparsamide.

DISCUSSION

DR. W. I. LILLIE, Philadelphia: I presume that every one is in accord that necessary precautions should be taken concerning tryparsamide therapy. About 6 per cent of patients treated with this drug have untoward ocular reactions, and by far the larger portion present only the transitory symptoms, which disappear shortly after the use of the drug is discontinued. In a small percentage a progressive loss of vision develops, and evidence of simple atrophy of the optic nerve may appear as a late or final result. Various procedures have been instituted to prevent such occurrences, and the ophthalmologist has played an important role in the early recognition of impending visual damage. This impending visual change is usually first suspected by subjective visual complaints, and if the use of tryparsamide is discontinued promptly, the subjective symptoms usually disappear.

The oral and intravenous use of various vitamins have been of great value in many systemic diseases. The vitamin B complex has been of real value in some of the diseases that affect the central and peripheral nervous systems. To my knowledge, the preliminary use of the vitamin B complex to prevent any untoward visual reaction during tryparsamide therapy has been suggested and first used by Dr. Muncy. The comparative statistics on his two groups of patients are suggestive that the vitamin B complex played an important role in decreasing and practically eliminating the occurrence of any untoward visual reactions. The subsequent improvement of vision in his group of patients who previously had been affected during the tryparsamide therapy is not so significant, as vitamin therapy has been used frequently, and the results obtained are uniformly good comparable to those in Dr. Muncy's group.

My experience with vitamin therapy for ocular conditions has been so uniformly favorable that I do not hesitate to use the combination of vitamin B and C, orally or intravenously, as an adjunct to other accepted types of treatment. Inflammatory conditions of the retina, choroid and

optic nerve respond equally well, and improvement in the retinitis of diabetes and scurvy is sometimes startling. The treatment of either the primary or the secondary type of atrophy of the optic nerve with vitamins or any other therapeutic measure is usually of no avail, but the progressive visual deterioration can sometimes be slowed up or eliminated. At least the use of vitamin B and C, orally or intravenously, can do the patient no harm and may be of benefit. Its use is an addition to present day therapeutic measures.

Dr. Muncy is to be congratulated for having originated the idea of using vitamin B therapy as a possible preventive of the untoward ocular effects arising during trypanamide therapy. I feel that ophthalmologists should establish a similar regimen, as suggested by Dr. Muncy, in their own institutions or practices, and later the sum total of individual experiences will answer Dr. Muncy's question: Does the preliminary use of vitamin B before trypanamide therapy is instituted lessen or eliminate the occurrence of untoward ocular effects?

DR. WILLIAM M. MUNCY, Providence, R. I.: I agree with all that Dr. Lillie had to say, but I should like to emphasize the following points: The 2 patients in the first series who had an adverse reaction but were again given trypanamide after they had been built up in health by a well balanced diet and had increased in weight were much slower regaining their lost visual fields than the later patients who were given vitamins intravenously. Furthermore, the failure in the last 2 cases was, I fear, due to overconfidence in the method being used.

The patient with atrophy of the optic nerve was given but 2 yeast tablets three times a day, and fever therapy was begun at once. Some writers hold that a high temperature dissipates vitamin B within the body. If so, the vitamin content of the body at this time was probably being decreased rather than augmented. Then to cap the climax, while the patient was still having high elevations of temperature, treatments with trypanamide were started. It was notable that the active use of vitamins intravenously rapidly restored the patient to his previous condition, and when the administration of trypanamide was again resumed, there was not a second reaction. If vitamin B complex is given by mouth, much larger doses should be used.

In regard to the patient who died, though his general condition had markedly improved and ocular examination showed as much advancement as one could expect, on going over the data since this paper was written it was discovered that other patients to whom trypanamide was again administered had all gained in weight while this patient had lost weight during his stay in the hospital.

While Dr. Lillie was discussing this paper, a number asked me if I gave yeast cakes. Yeast tablets are not yeast cakes but concentrated vitamin B complex obtained from grains that are put out by numerous drug firms.

Treatment of Accommodative Convergent Squint. DR. WILLIAM THORNWALL DAVIS, Washington, D. C.

If amblyopia ex anopsia is present in cases of accommodative convergent squint, occlusion of the fixing eye is necessary until the vision

is 20/40. The vision of the fixing eye may then be reduced to this level by partial occlusion with an opaque lens. Alternation must be avoided.

The child must be taught to keep his eyes straight with vision blurred, leaving his hypermetropic correction off as much as possible. The vision will clear as he learns to dissociate his convergence and accommodation. He must be taught to distinguish between clear and blurred vision. Blurred vision means that the eyes are straight, and clear vision means that one eye is turned in. The child should practice this with the orthoptist until he understands and gets the "feel" of knowing every time he turns his eye in. He must form the habit of keeping his eyes straight without glasses. When he can read 20/20 with both eyes at the same time, eyes straight, there is no longer desire or need for him to squint for distant vision. When he can read Jaeger's test type 1 or 2 with the eyes straight and without glasses, there is no longer any reason for squinting for near vision.

The mother or nurse should be taught to give the child lessons at home on a visual acuity chart, without glasses and for near and distant vision. Bar reading is prescribed first with the glasses, and when this can be achieved it is tried without glasses.

Exercises in the clinic or the office are given on the synoptophore, the diploscope and the Remy separator to teach the child to dissociate his convergence and accommodation, so that he can continue to do this after leaving the clinic. It takes an average of six months to one year to cure a patient of purely accommodative squint.

If there is a concomitant squint or other factor, such as hyperphoria or paresis of one of the ocular muscles, this factor obviously must receive appropriate treatment in addition to the treatment for the accommodative squint.

DISCUSSION

DR. GEORGE P. GUIBOR, Ottawa, Ill.: The ophthalmologists here are indebted to Dr. Davis for emphasizing in this paper the type of strabismus which is most amenable to nonsurgical treatment. This type, which is practically universally called "accommodative esotropia," is the type in which the deviation is less for distant than for near vision. It is improved by the wearing of convex lenses, usually a + 3 diopter sphere with a cylinder, by the use of atropine and by fusion training.

Dr. Davis has emphasized, too, that squint is a symptom complex and that several etiologic factors are associated with the strabismus in most cases. In a similar way he calls attention to what he considers a cure for strabismus. There should not only be a cosmetic recovery from the deviation but also a functional recovery. Thus the visual axis should be parallel for distant vision and for near vision, and the fusion power should be normal. As a matter of fact, however, few normal persons have perfect fusion without fusion training. One should, therefore, not expect every patient with parallel visual axis to have perfect fusion ability. Most of them, however, have good binocular vision but many times have imperfect stereoscopic ability.

Like all those interested in motor anomalies, Dr. Davis reiterates that the most important factor in securing a satisfactory recovery from the deviation is the diagnosis of the type of squint. He also discusses the diagnostic routine and here, too, impresses on his readers the importance

of determining the degree of deviation for distant and for near vision with and without corrective lenses. He again repeats these measurements when the eyes are under a cycloplegia and in the different directions of gaze.

One interesting sentence in this paper especially attracts my attention. This sentence is as follows: "As glasses are a physical, economic and social handicap, one must know that other means are available for the correction of the squint or for the relief of symptoms." This refers to accommodative esotropia. This sentence to me suggests that orthoptic fusion exercises and surgical treatment can be used in place of glasses in treating this condition. I have attempted this several times but have always felt that I was not doing the correct thing by stimulating convergence indirectly through the accommodation; I feel that this is a more difficult method of treating neuromuscular anomalies than when accommodation is kept under control by glasses and atropine. But I shall have to revise my attitude concerning this routine of removing lenses and attempting fusion exercises when such men as Dr. Davis and those of the London school of orthoptics follow this technic.

In conclusion, Dr. Davis emphasizes the following causes for failure in treating accommodative esotropia: (1) severe heterophoria or anomalous position of rest coexisting with the squint; (2) vertical imbalance; (3) visual defects, such as amblyopia, and (4) anomalous retinal correspondence.

The following cases, which are briefly reported, emphasize my limited experience with accommodative esotropia associated with some of these causes for failure. Of course pure accommodative esotropia never presented these complications.

One patient had right esotropia of 50 degrees without glasses and 40 degrees with glasses for distant and near vision. I thought that the condition was due to a spasm of the right internal rectus muscle, and to overcome that spasm I employed a horizontal prism, base out, in front of the right eye. These prisms improved the esotropia so that it was only 20 degrees after several months. I should like to ask Dr. Davis what he does when he attempts to treat a patient with a spasm of convergence or a spasm of one internal rectus muscle or a spasm of accommodation by his method. One of the methods I have used in the treatment of accommodative esotropia associated with a spasm of convergence is to employ a prism, undercorrecting, of course, the degree of spasm as determined by the occlusion or screen test.

In another case an anomalous position of rest was demonstrated. The patient had 30 degrees of left esotropia without his glasses and 25 degrees with his glasses. After both external rectus muscles were dissected, he had an apparent parallelism of his visual axes. However, the point to be brought out with regard to the anomalous position of rest is that, though I did not touch the internal rectus muscle, under cover the patient at the present time has an exotropia of 10 prism diopters, which was brought about by the mere resection of both external rectus muscles. Thus this patient had a convergent squint with a convergence insufficiency, a condition that the ophthalmologist often fails to recognize.

As a third example, the effect of eccentric fixation in anomalous retinal correspondence on the result of nonsurgical treatment is demon-

strated by 2 brothers. One was 7 years of age and the other 13. Both boys had 25 degrees of right esotropia with glasses. The deviations of these boys do not vary more than 5 degrees or more than 10 prism diopters. The other interesting factor is that both of them wear similar lenses before their eyes. The older boy, however, who has normal correspondence, recovered after one year; the younger, who has anomalous correspondence and eccentric fixation, did not recover.

Thus the most common factors which produce failures in treatment are: (1) severe heterotropia coexisting with the squint, (2) a vertical deviation, (3) visual defects and (4) anomalous correspondence.

DR. GRADY E. CLAY, Atlanta, Ga.: Dr. Davis has stressed the most important factor in the treatment of accommodative convergent squint, namely, orthoptics. The treatment of this type of squint has progressed little during the past decade. For years the refractive error alone was corrected. Later some ophthalmologists attempted to obtain a cosmetic result by surgical treatment. Only within the past twenty years have most ophthalmologists insisted on surgical treatment as soon as it has been seen that glasses would not correct the squint. There are a few bold enough to insist on such treatment before any attempt is made to help the patient with a correction.

It is true that the parents of a squinting child are primarily interested in a cosmetic result, but as an ophthalmologist one should be most interested in the development of binocular vision, and I should like to ask how one can be sure of ever obtaining fusion without the use of orthoptic training. It is true, and most unfortunate, that the ophthalmologists who discredit orthoptics are those who have not earnestly sought its true value.

Most persons who develop fusion with good amplitude of accommodation have very blurred vision at first, which clears with training, but, in my opinion, to start the treatment of such a person with the whole idea of removing the glasses is not good practice. For persons with accommodative squint and small errors, this may be done easily. The child with accommodative squint and a small astigmatic error who is given a +2 diopter sphere, which straightens the eyes, can be trained to develop fusion with good amplitude in a short period, and thus may in the course of time leave glasses off without discomfort. Many of the cases which Dr. Davis has reported are those in which a high hyperopic astigmatic error exists. I do not think his procedure should be attempted in such cases. If it is adopted, it seems to me that there is no reason to give a patient a correction. However, for girls it is most important that this be attempted, and I feel sure that the large majority of them can be trained because of their interest in trying to develop fusion and to hold their eyes parallel without glasses. For this particular type of person I would certainly recommend that the effort be made, with the hope that the patient may, for social purposes only, leave her glasses off.

DR. WILLIAM THORNWALL DAVIS, Washington, D. C.: For a subject as intricate as this, it is utterly impossible to give the many, many

details. As regards surgical treatment of accommodative squint, it should not be used for a purely accommodative squint. It is absolutely contraindicated in such cases. If operation is performed the squint will be corrected but the patient will have an exotropia for distant vision and an esotropia for near vision. Perhaps I am mistaken, but I have had this unfortunate experience a great many times. Such persons should not be operated on unless another factor is involved.

Dr. Guibor asked about spasm of extraocular muscles and accommodation. I have the feeling that a spasmodic action of one extraocular muscle must follow a paretic muscle and that there is another accommodative factor present. I may be wrong. My experience has been that it is dangerous, indeed, to operate in cases of anomalous retinal correspondence. In many cases the condition is incurable. If one disturbs the convergence system a wide swing in the opposite direction is likely to result and the patient will have a greater exotropia than esotropia, which cannot be corrected. I have learned that by bitter experience.

In regard to what Dr. Clay said about the use of glasses, I am entirely in sympathy with that. Of course, with high errors, particularly of astigmatism and spherical hypermetropia, the child will need glasses; but I was speaking of correction of the refractive error in regard to the squint only. I did not enter on the necessity of glasses for other purposes. Obviously, they may be necessary. What I meant to say was that one can cure accommodative squint associated with high refractive errors, so that it is not necessary for the patient to wear glasses to correct the squint. Perhaps I did not make that clear.

Of 98 persons with squint seen in the clinic of my associates and me from July 1938 to September 1939, 21 had accommodative squint. Seven are being treated now. The number whose treatment was incomplete due to insufficient cooperation is 6. The number considered corrected, that is, the number whose eyes are straight without glasses, is 8, or approximately 35 per cent. This is a rather low number, but one does not see many persons with purely accommodative squint. This is an important point. Of those included in the number whose squint was corrected, 3 had relapses two to three months afterward. It was necessary to institute active treatment again for these patients, and they responded promptly. Therefore, after treatment of accommodative squint a patient should be kept under observation for some time.

Visual Testing Methods in School. DR. JOHN B. HITZ, Milwaukee, Wis.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

Pulsating Exophthalmos. DR. SAMUEL J. MEYER and DR. H. SAUL SUGAR, Chicago.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

Retrograde Degeneration in the Optic Tracts and Nerves: An Experimental Study of Changes in the Axis-Cylinders.³ DR. P. J. LEINFELDER, Iowa City.

Previous experiments indicate that after section of the optic tracts little or no degeneration occurs in the myelin sheaths of the optic nerves. However, after section of the optic nerve, complete retrograde degeneration of the myelin sheaths is observed. The present study of the axis-cylinders was made to determine whether the reaction in the axis-cylinders was similar to that in the myelin. Experiments were made on cats and monkeys; the results of previous experiments were confirmed, and it was determined that after section of one or both optic tracts retrograde degeneration was not present in the axis-cylinders of the optic nerves. When the optic nerve was sectioned at the chiasm, there occurred complete retrograde degeneration of the axis-cylinders and myelin sheaths.

The study also shows that the extent of retrograde degeneration is not a phenomenon determined by the distance from the lesion, for when the optic tract was injured at, or remote from, the chiasm, degeneration proceeded in a similar degree to the chiasm.

Further studies are directed toward determining a connection between the optic chiasm and the hypothalamus in an attempt to explain this difference in degeneration.

Glaucoma Following Roentgen and Radium Therapy. DR. LOUIS BOTHMAN, Chicago.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

COLLEGE OF PHYSICIANS OF PHILADELPHIA,
SECTION ON OPHTHALMOLOGY

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Oct. 26, 1939

Rhinosporidiosis of the Conjunctiva: Report of a Case. DR. HAROLD J. BARNSHAW and DR. WILLIAM T. READ JR.

This article will appear in full, with discussion, in a later issue of the ARCHIVES.

Diplopia Following Fracture of the Malar Bone: Report of Cases. DR. H. MAXWELL LANGDON and DR. ROBERT H. IVY.

Diplopia has been frequently observed following fracture of the malar or zygomatic bone involving the floor of the orbit. Thus in 11 cases of fracture this sign was noted in 4 by one of us (R. H. I). In only 2 was

3. This work is a portion of research studies in neuro-ophthalmology being made in the department of ophthalmology of the University of Iowa under a grant from the American Academy of Ophthalmology and Otolaryngology.

a detailed study made of the diplopia. In both it was found to be due to paresis of the inferior oblique muscle (H. M. L.).

CASE 1.—W. S., aged 17, seen on April 10, 1934, had been struck on the left cheek by a baseball two hours before. There was ecchymosis of the tissues of the left side of the face with some conjunctival hemorrhage. The globe was uninjured. The following day the swelling had subsided after the use of ice compresses, and there was found to be definite diplopia when the patient looked upward—a typical sign of weakness of the inferior oblique muscle. Fracture of the floor of the orbit involving the malar bone was diagnosed. The following day the depressed bone was elevated by one of us (R. H. I.), and the diplopia disappeared permanently and has not been present since. Central vision was never disturbed, and the patient's eyes are in other ways perfectly healthy.

CASE 2.—Miss C. C., aged 25, fell on Dec. 21, 1938, striking the right maxillary region on the stone edging of a driveway. There were much contusion of the tissues, swelling of the lids and subconjunctival hemorrhage. Central vision was not disturbed and the eyeball was healthy; however, when the patient looked upward there was double vision typical of a weakness of the right inferior oblique muscle. A roentgenogram showed a fracture through the outer portion of the lower rim of the orbit. The patient was seen on December 25 and operated on by one of us (R. H. I.) on December 30. The next day the diplopia was slight and in a week had disappeared, since which time it has not returned.

Comment.—The remarkable feature of the diplopia in these cases was that it cleared up almost immediately after the fracture had been reduced. The mechanism of the disability and of the rapid recovery is difficult to explain. The diplopia could hardly be due to actual trauma to the muscle or to infiltration with blood or serum, because in that case at least several days would be required for recovery. If it were due to injury of the third nerve, recovery would be delayed still longer. If one looks at the anatomic relations one notes that the inferior oblique muscle arises from the anterior inferior angle of the orbit and has no other bony attachments but passes outward and backward in the floor of the orbit to be inserted on the outer surface of the eyeball. Its bony attachment is a considerable distance medial to the usual fracture line, so that the paralysis cannot be explained by tearing of muscle fibers at the point of origin.

In the literature the nearest approach to this subject is found in a recent article by Novick, who has reported 2 cases of paralysis of the inferior oblique muscle following the Caldwell-Luc operation on the maxillary sinus. He stated that paralysis or paresis of this muscle is exceedingly rare and that few ophthalmologists or otolaryngologists have observed a case of isolated paralysis of the muscle. In his cases, Novick attributed the paralysis to injury of the muscle by penetration by the curet of the thin orbital wall of the antrum in the region of the origin of the muscle.

In our cases this explanation does not apply, because there could hardly have been any tearing of the muscle fibers by the injury, and also there would not have been such prompt recovery from the diplopia after reduction of the fracture. The only explanation we can offer for this temporary diplopia is a sagging or loss of tension of the inferior oblique muscle, which was instantly relieved by reduction of the fracture. In all

of our recent cases of fracture of the malar bone with displacement the method described by Gillies has been used. It consists in the use of a long elevator introduced through a small incision in the skin above the hair line, beneath the temporal fascia and the zygoma. By this means the depressed bone can be readily elevated into position even two or three weeks after the injury, and the operation leaves no visible scar.

DISCUSSION

DR. CHAPIN CARPENTER: I should like to inquire of Dr. Ivy as to the patient I saw for him at the Bryn Mawr Hospital two weeks ago. The picture presented by this patient was exactly similar to that in the cases just reported. When I saw the patient it was about forty-eight hours after the operation, and he still had diplopia. He left the hospital the next day, and as I have not seen him since I have been wondering whether the diplopia cleared up.

DR. ROBERT H. IVY: I saw the patient mentioned by Dr. Carpenter this morning. I did not examine his eyes or test him for diplopia, but he said that the double vision had disappeared. The most important point about these cases is the necessity of early replacement of the depressed fragment in order to avoid permanent disfigurement. After three or four weeks the bone may become firmly fixed, and elevation is often impossible; replacement is usually simple if done in the first two weeks.

Management of Some Complications of Cataract Operations. DR. EDMUND B. SPAETH.

A series of complications following cataract extractions, including severe traumatisms, postoperative infections, subchoroidal hemorrhages, sympathetic ophthalmia, iritis with secondary glaucoma and prolapse of the iris are presented to illustrate certain important points in their treatment.

DISCUSSION

DR. FRANCIS HEED ADLER: This is an instructive paper, as one can usually learn more by hearing of the complications of cataract extractions than of the excellence of the results. The most annoying complication which I encounter is that of incarceration or prolapse of the iris. It does not seem to make much difference whether one does a complete or a peripheral iridectomy, prolapse sometimes occurs; when it is large it is difficult to handle.

I should like to ask Dr. Spaeth what his experience is with prolapse and if he knows of any method by which it can be prevented.

DR. WALTER I. LILLIE: Any one who performs operations on the eyes certainly encounters the complications that have been discussed by Dr. Spaeth. The use of sutures at operations for cataract minimizes the chances of severe prolapse of the iris. Also the use of a pressure bandage rather than a pad and a shield has, in my experience, lessened the occurrence of prolapse.

DR. ALFRED COWAN: I have examined a great many eyes after cataracts have been extracted, the operations having been performed by a number of surgeons. I think that there is hardly a case in which either prolapse of the iris or anterior synechia does not occur, regardless of the procedure used by the surgeon.

DR. LEIGHTON F. APPLEMAN: I have been wondering whether after the patient's eyes have been bandaged and he has been returned to bed he may not have a feeling of irritation which provokes squeezing of the orbicularis ciliaris in such a way as to produce pressure on the globe, causing the lips of the wound to open slightly and thus allowing the iris to slip forward and become caught between the lips of the wound. With this in mind, I have been particularly careful to warn the patients beforehand to avoid doing this during the time of healing.

DR. CARROLL R. MULLEN: With knowledge of the experimental work which had been done to prevent prolapse of iris, I performed a series of simple intracapsular cataract extractions with the use of corneoscleral sutures. Suturing consisted of a transverse passage through the cornea and a return transverse passage through the scleral or episcleral tissue, so that a double "lock" would close the operative incision. This was performed at the Wills Hospital. Instillations of solutions of paredrine (4-hydroxy- α -phenyl- β -aminopropane; β -4-hydroxyphenylisopropylamine) and homatropine hydrobromide were given an hour before the operation in order to obtain wide dilatation of the pupil. Intraocular tension was recorded previous to the use of this medication, immediately preceding the retrobulbar use of procaine hydrochloride and about two minutes after the latter injection. Care was taken that there was no prolapse before the patient left the operating room. A weak solution of physostigmine salicylate was instilled immediately after the operation in order to bring about more favorable contraction of the pupil. In my series of approximately 30 to 35 cataracts, I was dismayed with a 30 to 35 per cent occurrence of prolapse of the iris. The late operations were just as prone to this complication as were the early experiences. Needless to say, I have abandoned the simple extraction.

DR. LOUIS LEHRFELD: Many complications following cataract extractions may be prevented by careful study of the patient prior to operation. If the surgeon is aware of the fact that he has an uncooperative patient, he must be prepared to select anesthesia which will enable him to keep that patient under complete control during the operation. It makes no difference whether the anesthesia is general (ether or rectal anesthesia with avertin with amylene hydrate) or local (combination of procaine hydrochloride and cocaine hydrochloride). I feel that the complete anesthesia at the time of the operation will help considerably in preventing such complications as prolapse of the iris.

At the Wills Hospital I give routinely a retrobulbar injection of a 2 per cent solution of procaine hydrochloride, a subconjunctival injection of a 1 per cent solution of cocaine hydrochloride in the superior rectus muscle and another subconjunctival injection of cocaine hydrochloride at the point where the fixation forceps is applied to the eyeball. Local instillation of a 4 per cent solution of cocaine hydrochloride, as generally used by all surgeons, is likewise a part of the procedure to obtain complete anesthesia.

The patient is thus under complete control; in this way unnecessary and unexpected movements of the eyeball are avoided, making the cataract extraction possible without complications on the operating table. It also permits complete rest of the patient for several hours after the cataract extraction.

Corneoscleral sutures, as introduced by me at the Wills Hospital, have also served to prevent complications by permitting patients to get out of bed shortly or soon after the operation. I have had a number of patients who were permitted to sit in a rocking chair immediately on returning from the operating room and were allowed to go to bed only to sleep at night. In one particular instance, a stout woman, weighing approximately 300 pounds (135 Kg.), was so treated without complications. Other instances can be cited among the aged, who were treated in the same manner. Other patients are routinely permitted to get out of bed on the third, fourth or fifth day, depending on the individual circumstances. By this method the surgeon avoids the postoperative tympanites, backache, restlessness and tampering with the dressings.

Complete anesthesia of the eye and the use of corneoscleral sutures (or what I prefer to call corneoepiscleral sutures) are both instrumental in the prevention of postoperative complications.

DR. WILLIAM ZENTMAYER: Some reference has been made to the effect of bandaging on prolapse of the iris. I have not used a bandage after cataract operation for the last fifteen or twenty years, but have used instead narrow strips of isinglass plaster over the closed lid, running from brow to cheek, the only protection being a mask. The roller bandage which is applied around the head is objectionable. The disturbance of the bandage produced by movements of the head is transmitted to the pad in front and disturbs its position.

The only objection to the strips is that they cannot be made sterile, but I have never had infection from them. I do not know that my proportion of prolapses is any less than that of the average operator, but I feel that a bandage might be the cause in some of the cases.

DR. EDMUND B. SPAETH: There will be as many different decisions, opinions and recommendations in regard to this problem as there are ophthalmologists present. I cannot agree with some of those stated. In many instances sutures do prevent a prolapse of the iris, but prolapse does not occur until twenty-four to forty-eight hours after operation. The effects of the anesthesia are gone, the aqueous is reforming and anything that will interfere with the free passage of aqueous through the pupillary aperture from the posterior chamber into the anterior chamber will cause prolapse of the iris. It is for this reason that peripheral iridectomy is actually more efficacious in preventing prolapse of the iris than is complete iridectomy, for the peripheral iridectomy lies at the root of the iris. As Dr. Cowan said, the all too common finding of anterior synechia in the iridocorneal angle following complete recovery shows plainly the underlying cause in its formation. I know of no measure which can prevent it except that mentioned (the peripheral iridectomy and corneoscleral sutures) when preceded by an adequate toilet of the wound and by the all-important first forty-eight hours of uneventful convalescence.

Clinical and Experimental Studies of Interstitial Keratitis. DR. JOSEPH V. KLAUDER, DR. ELMER R. GROSS and DR. HAROLD F. ROBERTSON.

The following theories have been advanced for the motivating factor in interstitial keratitis: (a) that it is caused by direct invasion by

Spirochaeta pallida, (*b*) that it is due to an allergic reaction and (*c*) that it is due to a nutritional disturbance.

Sections of cornea removed from patients with interstitial keratitis failed to show *S. pallida* when examined by different methods.

Rabbits were given intraocular injections of horse serum. The ocular response to the second injection was studied by the slit lamp. There was no definite evidence of an allergic reaction. Pericorneal congestion and contraction of the pupil, however, occurred.

Cutaneous tests were performed on patients with interstitial keratitis, a piece of cornea of the same patient and also the cornea of a syphilitic fetus being employed. The reactions were negative.

It was not possible to produce interstitial keratitis in syphilitic rabbits by repeatedly traumatizing the cornea. The slit lamp picture of interstitial keratitis was produced in some normal and in some syphilitic rabbits by intracorneal injections of horse serum. This was attributed to trauma.

Studies were made of the effect of heat applied to the cornea in the treatment of interstitial keratitis. These embraced (*a*) the use of the thermophore (an instrument used to heat the cornea); (*b*) the measurement of the degree of heat penetration of the cornea of rabbits by the thermocouple; (*c*) the determination of the thermal death point of *S. pallida* in relation to the maximum temperature at which the cornea can safely be tested, and (*d*) the clinical effect of the thermophore in the treatment of the involved as well as the uninvolved eye of patients with interstitial keratitis and in the treatment of interstitial keratitis in syphilitic rabbits.

From these studies it was shown that the cornea is a poor conductor of heat. When the thermophore at 130 F. (54.4 C.) was applied to the anterior surface of the cornea, the maximum temperature of the posterior surface behind the thermophore was 111 F. (43.9 C.). The maximum temperature which the cornea will tolerate with safety is 130 F. (54.4 C.) for one minute. The temperature required to kill *S. pallida* was at least 120 F. (48.9 C.) for two minutes. It is therefore not possible after employing the thermophore in the treatment of interstitial keratitis to conclude by deduction that *S. pallida* are not present in the cornea.

Absorption of neoarsphenamine by the cornea after instillation into the conjunctival sac and the penetration of the drug into the cornea after intravenous administration in rabbits were studied. No arsenic was present in the cornea after successive instillation of neoarsphenamine in a dilution of 1:600. After six consecutive daily intravenous injections of neoarsphenamine, 30 mg. per kilogram of body weight, arsenic was present in each cornea in the amount of 0.04 mg.

The involved and uninvolved eye of patients with interstitial keratitis were treated with repeated instillations of 9 cc. of neoarsphenamine in a dilution of 1:600. This solution was maintained in the sac for twenty minutes. Such treatment did not influence the course of interstitial keratitis.

This article was published in full in the *Journal of Investigative Dermatology* (2:157-173 [Aug.] 1939).

Book Reviews

Primer of Allergy. By Warren T. Vaughan, M.D. Price, \$1.50. Pp. 140. with numerous illustrations. St. Louis: C. V. Mosby Company, 1939.

It is timely that Dr. Vaughan, who is well known for his large book entitled "Practice of Allergy," which is intended for the medical practitioner, should also undertake to write a primer in simple language for the lay patient.

The work is divided into a number of chapters, with a summary and questions and answers at the end of each chapter, which makes the subject matter comprehensible. The illustrations are amusing, as a number of the well known cartoons of Webster have been introduced. An important part is the last chapter on general orders and directions to the patient, which contains much necessary information.

ARNOLD KNAPP.

The Journal of Endocrinology. Price, \$6 a year. Published quarterly. London and New York, Oxford University Press.

This new journal will bring together the papers written by British investigators on endocrinology in one publication, which will be solely devoted to endocrinology. It is to be conducted by a council of management. Prof. E. C. Dodds is the editor. He is assisted by an editorial board composed of the following members: Dr. P. M. F. Bishop, Prof. C. D. Harrington, Prof. G. F. Marrian, Dr. A. S. Parkes, Dr. F. G. Young and Dr. S. Zuckerman. Dr. R. L. Noble has been appointed as assistant editor.

The first number appeared June 1, 1939, and contained eleven original articles. The journal will be published quarterly, and four issues will constitute one volume.

ARNOLD KNAPP.

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Place: Birmingham and Midland Eye Hospital.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
 Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
 Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
 Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.
 Place: Perth, Western Australia. Time: Sept. 2 and 7, 1940.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
 Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
 All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
 Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
 Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
 Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
 Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
 Time: July 4-6, 1940.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arie Feigenbaum, Abyssinian St. 15, Jerusalem.
 Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
 Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
 Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
 Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIEDADE DE OPHTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Francisco Ferreira, Pitangueiras 15, Brotas, S. Salvador, Brazil.
 Secretary: Dr. Adroaldo de Alencar, Brazil.
 All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
 Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7^e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
 Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arie-Friedman, 96 Allenby St., Tel Aviv, Palestine.
 Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON
OPHTHALMOLOGY

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
 Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
 Place: New York. Time: June 10-14, 1940.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY,
SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
 Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts
 Bldg., Omaha.
 Place: Cleveland. Time: Oct. 6-11, 1940.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
 Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
 Place: Hot Springs, Va.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
 Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
 Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.
 Place: Royal York Hotel, Toronto. Time: June 19-21, 1940.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
 Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.
 Executive Director: Mrs. Eleanor Brown Merrill, 50 W. 50th St., New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON
EYE, EAR, NOSE AND THROAT

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
 Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
 Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of
 each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Nadeau, 122 E. Walnut St., Green Bay.
 Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
 Place: The Gateway Inn, Land O'Lakes. Time: June 1940.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
 Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
 Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time:
 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick G. Sprowl, 421 Riverside Ave., Spokane, Wash.
 Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
 Place: Spokane, Wash. Time: June 24-27, 1940.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. Edward Clark, 1305-14th Ave., Seattle.
 Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
 Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. A. Shultz, 303 N. Main St., Rockford, Ill.
 Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
 Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
 Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
 Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIoux VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
 Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
 Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Dake Biddle, 123 S. Stone Ave., Tucson, Ariz.
 Secretary: Dr. M. P. Spearman, 1001 First National Bank Bldg., El Paso, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Wencke, Battle Creek.
 Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
 Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
 Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Virgil Payne, Pine Bluff.
 Secretary-Treasurer: Dr. Raymond C. Cook, 1005 Donaghey Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Melville Black, 424 Metropolitan Bldg., Denver.
 Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
 Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR,
 NOSE AND THROAT

President: Dr. Shirley H. Baron, 309 State St., New London.
 Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Grady E. Clay, 384 Peachtree St. N. E., Atlanta.

Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.

Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.

Place: Indianapolis. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.

Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Place: Davenport.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. D. C. Montgomery, 301 Washington Ave., Greenville, Miss.

Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Place: St. Charles Hotel, New Orleans. Time: April 25, 1940.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY

Chairman: Dr. O. B. McGillicuddy, 124 W. Allegan St., Lansing.

Secretary: Dr. A. R. McKinney, 330 S. Washington Ave., Saginaw.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Hendrie W. Grant, 330 Lowry Medical Arts Bldg., St. Paul.

Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.

Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Roy Grigg, Bozeman.

Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY,
OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. James S. Shipman, 542 Cooper St., Camden.

Secretary: Dr. Wright McMillan, 23 Passaic Ave., Passaic.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.

Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. G. Woodward, 100 S. James St., Goldsboro.

Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.

Secretary-Treasurer: Dr. F. L. Wicks, 516-6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. O. Clement, 406 State St., Salem.

Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.

Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
 Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
 Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m.,
 second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
 Secretary: Dr. J. W. Jervy Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
 Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg.,
 Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. J. W. Ward, 2607½ Lee St., Greenville.
 Secretary: Dr. Dan Brannin, Medical Arts Bldg., Dallas.
 Place: Fort Worth. Time: December 1940.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. A. E. Callaghan, Boston Bldg., Salt Lake City.
 Secretary-Treasurer: Dr. Rowland H. Merrill, 1010 First National Bank Bldg.,
 Salt Lake City.
 Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of
 each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
 Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd. S. W., Roanoke.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE
 AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
 Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
 Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
 Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Arthur G. Fort, 478 Peachtree St. N. E., Atlanta, Ga.
 Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
 Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month
 from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital,
 Baltimore.
 Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
 Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m.,
 fourth Thursday of each month from October to May.

BIRMINGHAM EYE, EAR, NOSE AND THROAT CLUB

President: Each member, in alphabetical order.

Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.

Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.

Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.

Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.

Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.

Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.

Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.

Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.

Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.

Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI OPHTHALMIC CLUB

Chairman: Each member, in rotation.

Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.

Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

CLEVELAND OPHTHALMOLOGIC CLUB

Chairman: Dr. Albert J. Ruedemann, Cleveland Clinic, Cleveland.

Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.

Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.

Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.

Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Harry M. Sage, 9 Buttles Ave., Columbus, Ohio.

Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.

Place: The Neil House. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.

Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.

Time: Second Friday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Dan Brannin, Medical Arts Bldg., Dallas, Texas.
 Secretary: Dr. L. E. Darrough, 4105 Live Oak St., Dallas, Tex.
 Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
 Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
 Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
 Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
 Time: 6:30 p. m., first Wednesday of each month.

DETROIT OPHTHALMOLOGICAL SOCIETY

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
 Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
 Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
 Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
 Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
 Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
 Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
 Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
 Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. J. Charles Dickson, 1617 Medical Arts Bldg., Houston, Texas.
 Secretary: Dr. William J. Snow, 708 Medical Arts Bldg., Houston, Texas.
 Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
 Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
 Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.

Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.

Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.

Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.

Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.

Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE AND EAR SOCIETY

President: Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.

Secretary-Treasurer: Dr. J. W. Fish, Brown Bldg., Louisville, Ky.

Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St. N. W., Washington.

Secretary: Dr. Elmer Shepherd, 1606-20th St. N. W., Washington.

Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.

Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Herbert G. Smith, 411 E. Mason St., Milwaukee.

Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.

Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.

Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.

Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.

Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St. W., Montreal, Canada

Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, 119-7th Ave., Nashville, Tenn.

Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.

Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW HAVEN OPHTHALMOLOGICAL SOCIETY

President: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. David H. Webster, 140 E. 54th St., New York.
Secretary: Dr. Robert K. Lambert, 10 101-15th Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Arthur M. Yudkin, 257 Church St., New Haven, Conn.
Secretary: Dr. Benjamin Esterman, 515 Park Ave., New York.
Place: Squibb Hall, 745-5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND
OTO-LARYNGOLOGICAL SOCIETY

President: Dr. J. T. Maxwell, 1140 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner;
7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Adolph Krebs, 509 Liberty Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351-5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. J. F. Hardesty, Missouri Theatre Bldg., St. Louis.

Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.

Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.

Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.

Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE,
EAR, NOSE AND THROAT

Chairman: Dr. Matthew Hosmer, 384 Post St., San Francisco.

Secretary: Dr. Fred Boyle, 490 Post St., San Francisco.

Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. I. Henry Smith, Slattery Bldg., Shreveport, La.

Secretary-Treasurer: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.

Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter W. Henderson, 407 Riverside Ave., Spokane, Wash.

Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.

Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.

Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.

Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.

Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.

Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. Ernest Sheppard, 927 Farragut Sq. N. W., Washington, D. C.

Secretary-Treasurer: Dr. E. Leonard Goodman, 1801 I St. N. W., Washington, D. C.

Place: Episcopal Eye and Ear Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

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WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

Chairman: Each member in turn.

Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.

Place: Office of chairman. Time: Last Tuesday of each month from October to May.

CONCRETIONS IN A LACRIMAL CANALICULUS CAUSED BY ACTINOMYCES

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AMSTERDAM, NETHERLANDS

Concretions due to actinomyces may occur in the superior or in the inferior canaliculus.

If the inferior canaliculus is involved, the first symptom is epiphora. There may be a slight swelling in the region of the canaliculus and dilatation of the punctum lacrimale (fig. 1). In early stages, however, these signs may be absent or so slight that they may be easily overlooked and a diagnosis of stenosis of the lacrimal passages made. On syringing the tear sac, there is no interference with the passage of fluid to the nose. On pressure in the region of the canaliculus, a droplet of pus appears at the punctum. In later stages the swelling of the canaliculus is noted at first sight, the ectasia of the canaliculus being considerable (fig. 2).

If the superior canaliculus is affected, there are no early symptoms, since an obstruction of the superior canaliculus does not cause epiphora. In later stages the physician may make a diagnosis of chalazion (which does not occur in this region), atheroma or inflammation, or a tumor may be suspected. In a case in which tumor was thought to be present (fig. 3), pressure caused a drop of pus to appear at the superior lacrimal punctum (fig. 4). The canaliculus was slit open. It contained several clumps of soft yellowish material, which did not adhere to the wall (fig. 5).

In the first case I saw, in 1933, the organism was isolated and studied by Dr. Ruys, bacteriologist.¹ It proved rather difficult to obtain the pure culture, which is necessary for a correct differentiation between *Lep-
tothrix* (simple filaments) and *Actinomyces* or *Streptothrix* (branches).

The following technic, which I used on the advice of Dr. Ruys in 2 other cases, is so simple and efficient that it enabled me to isolate the organism in the laboratory of the ophthalmic hospital.

From the Department of Ophthalmology, University Hospital.

1. Ruys, A. C.: Concretions in a Lacrymal Canaliculus Caused by *Actinomyces*, *Brit. J. Ophth.* **19**:385, 1935.

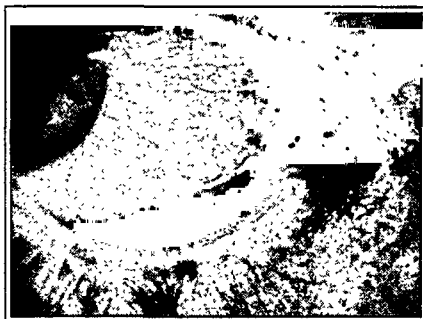


Fig. 1.—Early involvement of the inferior canaliculus produced by *Actinomyces*. There are slight swelling of the canaliculus and dilatation of the punctum lacrimales.



Fig. 2.—Later stage of the condition, showing considerable ectasia.



Fig. 3.—Involvement of the superior canaliculus. In this case the appearance caused the presence of a tumor to be suspected.



Fig. 4.—Appearance of pus at the superior lacrimal punctum on pressure in the region of the canaliculus.



Fig. 5.—Clumps of soft yellowish material found in the pus.

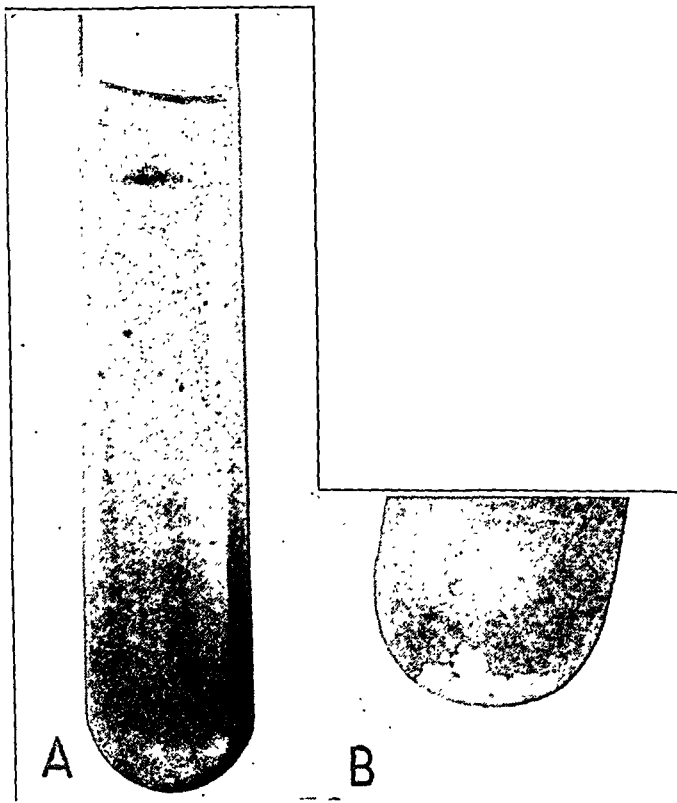


Fig. 6.—*A*, pure culture of *Actinomyces* grown in dextrose agar. *B*, a secondary culture grown in dextrose broth.

After the ducts were slit, a few concretions were isolated and a drop of 96 per cent alcohol put on each of them. After evaporation of the alcohol, which killed the bacteria, an emulsion was made of this material in sterile physiologic solution of sodium chloride. A test tube with sterile dextrose agar was put in a hot water bath until the medium became liquid. Then the temperature was allowed to drop again. Just before the agar became solid, a number of drops of the emulsion was added and the tube rolled between the hands in order to mix the material with the dextrose agar. The tubes were put in an incubator at 37 C. By this method it was easy to obtain a pure culture, which grew especially at a certain level under the surface of the dextrose agar (fig. 6 *A*). A secondary culture of material from this region grew in dextrose broth in the form of crumblike masses at the bottom, the fluid remaining perfectly clear (fig. 6 *B*). In some instances it was possible to get a pure culture without using alcohol simply by repeated washing of the concretion in physiologic solution of sodium chloride.

This method enables the ophthalmologist to obtain a pure culture. Further investigation of this interesting organism is possible only by a bacteriologist who has made a special study of the difficult field of Actinomyces.

SUMMARY

The clinical picture produced by Actinomyces in the superior and the inferior canaliculus is described, and a method is given for obtaining a pure culture of the organism.

TREATMENT OF OCULAR SYPHILIS

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AND

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Syphilis is a chronic infectious disease caused by *Spirochaeta pallida*, which invades all the tissues of the body. No organ is immune to the possible effects of the invasion. The manifestations of the disease may appear at any time as long as the micro-organism is active. It is the dermatologist who usually sees the first and most evident signs of the fresh infection and who is in a position to treat the disease possibly before the development of serious internal complications. The eye may be involved in the earliest stage of the infection. In ocular involvement vital and delicate tissues are affected.

Today the study of the eye is never neglected during the examination of the patient. Irregularity and "baying" of the pupil may be the first signs indicating possible syphilitic infection. Plastic iritis may occur in the early months of syphilis. Neuroretinitis may be observed early in the disease and may even disappear without treatment. It may, however, run an insidious course and remain dormant for years or appear as a late manifestation. Congenital interstitial keratitis is fairly common during the first few years of life and tends to resist treatment. The eyegrounds and the oculomotor apparatus demand frequent and repeated examinations in order to detect the possible presence of involvement of the nerve through basilar disturbance or pressure.

The ideal method of treatment calls for close cooperation between the ophthalmologist and the syphilologist. In the usual case the lesions of syphilis respond to regularly recognized methods of procedure, as, for instance, extragenital chancre of the conjunctiva, which would be treated in similar fashion to hunterian lesions elsewhere. Other manifestations, such as syphilids of the eyelids, gummatous infiltrations of the orbit, gummatous episcleritis and chorioretinitis, usually respond to the appropriate therapy employed for the early or late stages of the disease. As has already been indicated, in most cases there are no special procedures, ophthalmic or otherwise, to be adopted except in the presence of certain complicating features.

Read before the New York Society for Clinical Ophthalmology, Oct. 2, 1939.

From the Department of Dermatology and Syphilology, Mount Sinai and Beth Israel Hospitals.

AVAILABLE DRUGS

In order to prevent the serious ocular complications of syphilis, it is most imperative that the disease be recognized early and immediate treatment instituted. Continuous treatment should be given to prevent relapses, which are more apt to occur when treatment is interrupted with rest periods. The end results justify continuous treatment for years. The physician who administers the treatment must, however, be cognizant of the good and ill effects of the various remedies which he employs.

Arsphenamine still remains the most potent antisyphilitic drug. Mercurial preparations, at one time the standard remedy, are inferior to compounds of arsenic and of bismuth as a spirocheticide. Bismuth preparations and the newer arsenicals, while of great value, are still inferior to the arsphenamines. The average dose of arsphenamine is 0.4 Gm. for men and 0.3 Gm. for women; the drug is administered intravenously, usually in courses of six to eight weekly injections, coincident with or alternating with courses of compounds of the heavy metals.

For a long time the ophthalmologist feared the action of the arsenicals because of toxic reactions from sodium arsinate, which had produced amblyopia in numerous cases. Again, the development of atrophy of the optic nerve in cases of neurosyphilis in which tryparsamide therapy was employed also delayed the progress which had been made in the treatment of ocular syphilis with arsenicals. It is now known that tryparsamide when properly given as indicated is most valuable in the treatment of neurosyphilis and will rarely produce irritation of the optic nerve when contraindications are excluded. Although it has been shown that tryparsamide is without great spirocheticidal action generally, it has strong penetrating powers for the tissues of the central nervous system. It is of most value in the treatment of ocular syphilis associated with dementia paralytica of the tabetic form. The average dose is 1 to 3 Gm. administered every second week for at least twenty weeks.

Sulfarsphenamine is a sulfonate derivative of arsphenamine containing 20 per cent arsenic. It can be injected intramuscularly rather than intravenously. This advantage is outweighed by the high frequency of reactions it produces, and its use is now confined to children, who apparently tolerate it better than adults. The average dose is 0.6 Gm. for men and 0.4 Gm. for women. Silver arsphenamine is another arsphenamine compound; it contains 20 per cent arsenic and 13 per cent silver. It is considered by some syphilologists to be of value in the treatment of early visceral disease and neurosyphilis. The dose is approximately 0.2 to 0.3 Gm., and the drug is given intravenously.

Mapharsen, an arsenoxide, although less efficacious than arsphenamine, is a valuable drug in doses of 0.02 to 0.06 Gm. for those persons who are sensitive to the arsphenamines. Combinations of bismuth and arsenic, such as bismarsen, are being used with more frequency because the intramuscular route may be employed. Bismarsen contains 13 per cent arsenic and 23 per cent bismuth, and its action is slower than that of other arsphenamines. However, the results with bismarsen in the treatment of early syphilis are satisfactory if the injections are given at intervals of three to four days in doses of 0.1 to 0.2 Gm.

Acetarsonne has shown good therapeutic action in some cases of neurosyphilis, but the high incidence of toxic reaction after its use, particularly nephritis, is a serious drawback.

Finally, it is emphasized that the use of iodides and their most beneficial effects should not be forgotten in the battle against syphilitic invasion. The average dose is approximately 5 to 10 Gm. daily.

Within the past few years the administration of large doses of arsphenamine by intravenous drip has been attempted. Hyman and his associates¹ have employed the gravity method of intravenous therapy and have administered as much as 1 Gm. of neoarsphenamine every fifteen hours in a 5 per cent dextrose solution. A total of about 5 Gm. is usually given. While this method yields immediate clinical and serologic results that are good, the toxicity, as manifested by the frequent occurrence of febrile reactions, toxicodermas, neuritides and fatal hemorrhagic encephalitis, places the method within the experimental stage. In addition, the possibility of reinfection is a drawback, as the immunity developed may not last for more than a few months or years. For the treatment of ocular syphilis the method of most value is still continuous therapy with bismuth compounds and arsphenamine.

Schereschewsky² advocated quinine for treatment-resistant syphilis, especially when the disease is associated with parenchymatous keratitis. He advised a dose of 2 Gm. daily for four days in addition to arsenotherapy. We have had no experience with quinine in this condition.

NONSPECIFIC THERAPY

Various methods of nonspecific antisyphilitic therapy may be employed to raise the body temperature to at least 104 degrees F. The fever may be produced by injections of malarial parasites, typhoid vaccine and sterile milk as well as by physical measures, such as hot

1. Hyman, H. T.; Chargin, L.; Leifer, W., and Rice, J. L.: Massive Dose Chemotherapy of Early Syphilis by the Intravenous Drip Method, *J. A. M. A.* **113**:1208 (Sept. 23) 1939.

2. Schereschewsky, J.: Therapieresistente Syphilis insbesondere die Keratitis parenchymatosa und ihre Behandlung mit Chinin, *Klin. Wchnschr.* **14**:381, 1935.

baths, electric blankets, heat cabinets, diathermy, electric induction coils and the inductotherm.

Nonspecific therapy plays an important role when the arsenicals and the heavy metals have failed. It has been shown that spirochetes in man are killed by a tissue temperature of 42 C. for one hour. Various hypotheses have been offered to explain the biologic reactions which occur when the fever is produced. Fever therapy apparently produces an immunologic response from the reticuloendothelial system.

It is believed that the high temperature tends to destroy the spirochetes and resorb the lymphocytic infiltrations of the lesions. It is also claimed that the reticuloendothelial system is stimulated to produce dereagins, which are carried to the inflammatory zone, where they attack and weaken the spirochetes. It is therefore of great value to keep the blood vessels patent by iodides administered orally in order to facilitate transportation of these reagins to the involved sites.

Hyperpyrexia will at times benefit atrophy of the optic nerve, keratitis, uveitis and pupillary lesions when specific therapy had previously failed. The response in cases of syphilitic interstitial keratitis is most noteworthy.

The ill effects of hyperpyrexia may be reduced by the preliminary administration of fluids, saline solution, dextrose and calcium. Patients with early involvement and minimal damage to the tissue receive the best results from treatment. Reenforcement by chemotherapy enhances the possibility of a good effect. The results from fever therapy are better when preceded by one or two courses of treatment with arsphenamine and a bismuth compound.

THERAPY OF SPECIFIC SYPHILITIC OCULAR LESIONS

Interstitial Keratitis.—This common lesion of congenital syphilis is resistant to treatment. Prophylactic therapy of early syphilis does not always prevent the later occurrence of interstitial keratitis. However, if prophylaxis can be extended to include the antisymphilitic treatment of the pregnant woman, the incidence of congenital syphilis, and accordingly of interstitial keratitis, would be markedly decreased. At one time it was believed that keratitis was a self-limited disease, and treatment was symptomatic. The modern trend, however, is definitely in the direction of active therapy. Carvill and Derby,³ in a large and well studied group of cases described excellent results in 74 per cent of the cases in which treatment was given and in 38 per cent of the cases in which therapy was not administered, with a 3.6 per cent incidence of relapse in the former group as contrasted with 27 per cent in the latter group.

3. Carvill, M., and Derby, G. S.: *Interstitial Keratitis*, Boston M. & S. J. 193:403; 1925.

Moore⁴ advocated continuous therapy over a two year period consisting of alternating courses of arsphenamine and a bismuth compound. He claimed that most failures are due to underdosage. In some instances a Herxheimer reaction may be noted but is rarely of importance. Potassium iodide should be given simultaneously (3 to 7 Gm. daily) at the onset. Disappearance of the lesion proper may occur within twelve weeks, and further therapy may even lead to resolution of the usual residual corneal opacity. Schamberg and Wright⁵ reported good results with alternate injections of a bismuth compound and sulfarsphenamine. Occasionally, cases will be encountered in which the disease is resistant to treatment, and a modification in therapy is advisable. The substitution of different arsenicals or bismuth preparations will frequently effect a cure in the cases in which the syphilis is recalcitrant. Beneficial results have been obtained by injection of foreign protein. Menagh⁶ reported 4 cases of interstitial keratitis, in 3 of which the syphilis cleared after the addition of hyperpyrexia to chemotherapy. In 2 of these cases relapse occurred on several occasions prior to the institution of fever therapy.

Fever therapy tends to reduce the course of the disease and relieves the acute symptoms of pain, photophobia, blepharospasm and excessive lacrimation. Löwenstein⁷ highly praised the value of roentgen rays, especially for patients with resistant corneal opacities. For those persons who respond poorly to intensive therapy, his method offers another weapon in the therapeutic armamentarium. Corneal transplantation is still another. Locally, atropine, ethylmorphine hydrochloride and hot compresses are useful early; in the retrogressive stage, yellow mercuric oxide is valuable.

Atrophy of the Optic Nerve.—The primary type of atrophy of the optic nerve observed in cases of cerebrospinal syphilis, tabes and dementia paralytica is fortunately not of frequent occurrence. If the condition is untreated, it leads to complete blindness. After blindness has occurred, obviously no replacement of the dead fibers can be accomplished. When the atrophic process is still unilateral and visual acuity and fields in the better eye are nearly normal, the condition may be aborted. When there is simultaneous bilateral loss of visual acuity and constriction of form and color fields, the prognosis is relatively bad.⁴ There are two types

4. Moore, J. E.: *Modern Treatment of Syphilis*, Springfield, Ill., Charles C. Thomas, Publisher, 1933, p. 328.

5. Schamberg, J. F., and Wright, C. F.: *The Treatment of Syphilis*, New York, D. Appleton and Company, 1932.

6. Menagh, F. R.: *The Treatment of Syphilis with Hyperpyrexia*, *Am. J. Syph.* **21**:609, 1937.

7. Löwenstein, A.: *Zur Pathogenese und Therapie der Keratitis parenchymatosa*, *Klin. Monatsbl. f. Augenh.* **88**:306, 1932.

of treatment, the Swift-Ellis method of intraspinal arsphenamine therapy and hyperpyrexia. Intraspinal treatment is administered at intervals of two weeks in courses of seven to ten injections. It should be followed by a course of injections of bismuth compound and then repeated. Obviously, this is a very technical treatment, to be administered only by experts. The intracisternal route may also be employed to advantage. Combined subdural and intravenous therapy may lead to therapeutic shock, but the risk is far outweighed by the possibility of arresting inevitable blindness. Treatment should be continued for over six months after progression of the lesion has ceased.

The utilization of fever therapy is primarily reserved for those cases of atrophy of the optic nerve associated with paresis or dementia paralytica of the tabetic form.⁴ Clark⁸ reported an encouraging series of cases in which benefit was obtained in 8 of 12 cases after malarial therapy. Intramuscular injections of sulfur have also been used to produce high temperatures. Menagh⁶ used hyperpyrexia in 10 cases of atrophy of the optic nerve. The disease in 3 was of long standing and showed no improvement. Four patients did well, 2 did poorly and 1 discontinued therapy. Menagh concluded that if there is damage due to a localized cellular infiltrate, as in tabes, the disease will respond to fever therapy and to early treatment. In dementia paralytica, on the other hand, the process usually spreads from the brain to the optic nerve and is then but part of a generalized and deep-seated infection with a poorer prognosis. In a similar vein, Culler and Simpson⁹ stated that ocular syphilis due to exudative lesions should be expected to respond to fever therapy. Hargraves and Doan¹⁰ demonstrated that artificially induced fever destroys lymphocytes. This decrease in perivascular infiltration of lymphocytes may account for part of the therapeutic effect, plus the direct thermolethal effect on the spirochetes. The results reported by 11 different observers for 98 patients with primary atrophy of the optic nerve treated by fever therapy showed 39 per cent improvement as contrasted with 54 per cent improvement obtained by Moore with subdural treatment.

Tryparsamide should be mentioned at this point because of its value in the treatment of neurosyphilis. It is to be used in the presence of atrophy of the optic nerve only when the patient has complete loss of vision.

8. Clark, C. P.: Role of Malaria in Control of Atrophy of the Optic Nerve Due to Syphilis: Study of Twelve Cases, *Arch. Ophth.* **15**:250 (Feb.) 1936.

9. Culler, A. M., and Simpson, W. M.: Artificial Therapy in Cases of Ocular Syphilis, *Arch. Ophth.* **15**:624 (April) 1936.

10. Hargraves, M. M., and Doan, C. A.: The Physiological Response of the Hemopoietic Tissues to Artificially Induced Fever, in *Abstracts of Papers of the Fifth Annual Fever Conference*, Dayton, Ohio, 1935.

Lauber¹¹ advanced the theory that all of the drugs commonly used in the treatment of syphilis (arsphenamine, mercury and bismuth compounds and iodides) raise the systemic blood pressure and secondarily the retinal blood pressure. This, in his opinion, is the reason for the deleterious effects of antisyphilitic therapy in primary atrophy of the optic nerve. Therefore, he attempted to treat a series of patients by lowering the intraocular tension, since it was impossible to lower the systemic blood pressure. He treated 33 patients with a solution of a pilocarpine salt in 2 per cent or higher concentration, and if this was ineffective, he employed cyclodialysis. These two procedures led to a lowering of intraocular tension to a level of 10 to 14 mm. of mercury. In 27 of 33 patients, improvement was noticed. Unfortunately, the duration of the observation was not stated, and the results cannot be held as conclusive.

The Uveal Tract.—Involvement of the uveal tract and its component structures, notably, the iris, usually occurs in late secondary syphilis. Stokes¹² stated that 40 per cent of iritis is syphilitic and that iritis constitutes 73 per cent of the total ocular complications of early syphilis. It is commonly associated with a relapsing early syphilitic process. It is not the rule for iritis to occur as an isolated lesion but as a forerunner of more serious optic involvement, such as neuritis of the nerve or neuroretinitis. It is toward this complication as well as toward the iritis itself that therapy should be directed. The treatment should consist of alternating courses of arsphenamine and a bismuth compound with iodides by mouth. Therapy is continuous, as in early syphilis, with overlapping of the courses of arsphenamine. This is important, because a few days without treatment may be dangerous in the interim periods and may precipitate a relapse. It should be stressed that healing of the iritis is not an indication for cessation of treatment. Therapy must be continued for at least one year after the blood has given a negative serologic reaction, and the blood must be maintained persistently in this state. When iritis occurs as a feature of late syphilis, here again the stage of the disease is treated rather than the local manifestation. At this time, therapy should consist of at least two years of continuous treatment followed by at least two or more years of intermittent therapy. The drugs of choice are the arsphenamines and bismuth preparations. Cole and his associates¹³ follow up this method with administration of compounds

11. Lauber, H.: Treatment of Atrophy of the Optic Nerve, *Arch. Ophth.* **16**: 555 (Oct.) 1936.

12. Stokes, J. H.: *Modern Clinical Syphilology*, Philadelphia, W. B. Saunders Company, 1934.

13. Cole, H. N.; Moore, J. E.; O'Leary, P.; Stokes, J. H.; Wile, U. J.; Clark, T.; Parran, T., and Usilton, T. J.: The Clinical Outcome of the Treatment of Latent Syphilis, *Ven. Dis. Inform.* **13**:351 and 371, 1932.

of the heavy metals for as long as ten years after discovery of the syphilitic process. Bismuth preparations are undoubtedly of greater value than mercurials in these cases and are less toxic.

The local treatment of iritis resolves itself into the employment of atropine and its derivatives for the dilatation of the pupil with the intent of breaking any adhesions which may have formed between the lens and the iris. Rest in bed and the administration of salicylates for relief of pain round out the therapy. Gieske and Moore¹⁴ reported good results in 87 per cent of cases of early syphilitic iritis and practically no residual damage in the iris in 58 per cent of cases of late syphilis. The occurrence of uveoparotitis in association with syphilis has been noted, and, according to Folger,¹⁵ many forms of therapy have been tried, but none has proved of any real value.

Chorioretinitis.—This lesion may occur in congenital syphilis prior to the onset of interstitial keratitis but is more frequently a complication of acquired syphilis. The appearance of this lesion has been noted during treatment with a compound of one of the heavy metals other than arsphenamine, and in 1 case described by Zimmermann¹⁶ six relapses were noted. As these lesions do not occur during arsenical therapy, longer courses of arsphenamine are advised with a suggested change from the arsenical first employed to a slightly different arsenic compound. Foreign protein therapy as well as hyperpyrexia may also be of benefit and may be utilized alone or in conjunction with chemotherapy. The chorioretinitis of congenital syphilis runs a comparatively mild course, and its treatment is that of the stage of the disease proper.

Dacryocystitis.—Involvement of the lacrimal sac may occur in about 2 per cent of the patients with congenital syphilis. The condition is usually unilateral, and the treatment is essentially surgical.

COMPLICATIONS OF AND REACTIONS TO THERAPY

Tryparsamide.—The utilization of this drug in cases of neurosyphilis has again contributed to the ophthalmologist's distrust of the arsenical group. Tryparsamide has a definite sphere of usefulness in the treatment of neurosyphilis but should not be employed when the optic nerve or retina is involved. Sloan and Woods¹⁷ recently reviewed the literature and collected reports of 2,087 cases in which tryparsamide was administered. Six and three-tenths per cent of the patients had subjective

14. Moore, J. E., and Gieske, M.: Syphilitic Iritis, *Am. J. Ophth.* **14**:110, 1931.

15. Folger, H. P.: Uveoparotitis (Heerfordt): Report of a Case, *Arch. Ophth.* **15**:1098 (June) 1936.

16. Zimmermann, E. L.: Syphilitic Iridocyclitis with a Consideration of Factors Influencing Its Occurrence, *Arch. Ophth.* **53**:549, 1924.

17. Sloan, L. L., and Woods, A. C.: The Effect of Tryparsamide on the Eye, *Am. J. Syph.* **20**:583, 1936.

reactions and 3.5 per cent had objective complaints. They stated that there are two types of reaction. The first is the acute and less common type. It consists of the development of almost complete blindness within several days after the first few injections. The second type is decidedly more common and is chronic. It consists of contraction of the visual fields with retention of normal central vision and the absence of objective signs of atrophy of the optic nerve. They observed that in patients with normal optic nerves, fields and vision prior to tryparsamide therapy, there is little danger of any serious ocular damage if the administration of the drug is permanently discontinued at the first appearance of defects of visual fields. An occasional temporary, alarming but not serious untoward reaction is that of a nitritoid character appearing usually after several injections have been administered.

Arsphenamines.—That the arsphenamines are still the most potent antisyphilitic remedies at one's disposal is no longer a disputed subject. Their reputed toxic effects on the ocular apparatus have been disproved by the relative infrequency of reactions in thousands on thousands of cases. Zimmermann¹⁸ has subdivided the toxic effects of the arsphenamines on the eye into three groups.

True Toxic Reactions: The only true toxic arsphenamine reaction involving the normal eye occurs during the nitritoid crisis. This general congestive reaction includes flushing of the face, swelling of the lips and eyelids, profuse perspiration, pallor, dyspnea, rapid pulse, vomiting, suffocation and even death. In addition, the eye exhibits hyperemia of the conjunctiva. This reaction may occur during or immediately after injection. The administration of the arsphenamine should be stopped immediately, and, if necessary, epinephrine administered. Other optic structures, especially the optic nerve, are never affected by arsphenamine. A secondary, but rare, toxic ocular complication occurring usually in the presence of an arsenical exfoliative dermatitis is superficial keratitis or corneal ulcer.⁴

The Jarisch-Herxheimer Reaction: This name is given to an efflorescence of the local syphilitic manifestations, which occurs after the first few injections of arsphenamine. It is usually a phenomenon of secondary syphilis, but it may complicate late syphilis. Zimmermann¹⁸ expressed the belief that the Herxheimer reaction was of clinical importance only in patients with optic neuritis or primary atrophy of the optic nerve. He cited a case of his own and several others culled from the literature in which there occurred a violent hemorrhagic exacerbation of a preexisting optic neuritis with permanent visual damage after a single large dose of arsphenamine. He also has noted sudden complete loss of

18. Zimmermann, E. L.: The Role of the Arsphenamines in the Production of Ocular Lesions, *Arch. Ophth.* 57:509, 1928.

vision in patients with primary atrophy of the optic nerve after the institution of arsenical therapy. He advised the administration of a heavy metal compound first to this type of patient or the use of very small doses of the arsenicals. Moore⁴ stated that these reactions must be rare as he has never seen them. He expressed the opinion that he did not believe it necessary to employ long preparatory treatment with compounds of the heavy metals, especially cases of atrophy of the optic nerve. For optic neuritis, he advised starting treatment with small doses (i. e., 0.1 Gm. of arsphenamine) but increasing the dose rapidly at five day intervals to an average amount. Moore further observed that while the normal eye is unaffected, the diseased eye may be seriously injured by a Herxheimer reaction. This may appear either as an intensification of an already existing lesion, as a flare-up in a quiescent lesion or as the unexpected appearance of an inflammatory process in structures previously apparently uninvolved. According to Stokes,¹² one third of the practitioner's troubles comes from the nitritoid reaction.

Ocular Relapses: This effect is a result of insufficient arsphenamine therapy. The optic lesions may occur as part of a neurorecurrence or as isolated phenomena. The most common types are optic neuritis and iridorelapse. The probable mechanism is the destruction by insufficient arsenical therapy of all the spirochetes with the exception of those lurking in the depths of the eye or the far corners of the central nervous system. After an incubation period of several months exacerbations occur in these regions and rear their ugly heads as neurorecurrences. The only appropriate form of treatment is the immediate institution of intensive arsenical therapy. Dreyfus¹⁹ recommended four to six courses of arsphenamine, each consisting of 8 to 10 Gm. of the drug. Treatment is continued until the tests of the blood and spinal fluid have become negative and have remained so for one year. Periods of rest from treatment are definitely not permitted.

Errors in Treatment.—Kazas²⁰ stated that there are five errors in the treatment of ocular syphilis. These are mentioned with a view to thoroughness, although complete agreement is not implied.

(a) Commencement of therapy with bismuth or arsphenamine instead of mercury may lead to biotrophic reactions with undesirable complications.

(b) The provocative test for diagnosis may produce a temporary thriving of the spirochetes.

19. Dreyfus, G. L.: *Spezifische und unspezifische Tabestherapie*, Deutsche Ztschr. f. Nervenhe. **84**:14, 1925.

20. Kazas, I. I.: *Five Errors in the Treatment of Syphilis of the Eye*, Sovet. vestnik oftal. **7**:230, 1935.

(c) Insufficient dosage due to fear of toxicity of arsphenamine is a third error. A knowledge of the essential pharmacologic effects of arsphenamine, i. e., vagotonicity and acidophilia will avoid this.

(d) Monotherapy is a mistake.

(e) Inability to distinguish between insufficient dosage of arsphenamine and the effects due to overdosage is the final error.

Kazas treats his patients with injections of a soluble mercurial every day for the first two weeks. He then follows with arsphenamine, a bismuth compound and the iodides. His basic rule is the administration of as much arsphenamine as the patient can tolerate.

SURGICAL TREATMENT, ITS INDICATIONS AND CONTRAINDICATIONS

The influence of trauma in the development of sites of elective localization in syphilis is well known. The development of gummatous infiltrations in the healing scars of syphilitic wounds has been noted in many instances. The unfortunate features of this complication are its utter unpredictability and its usual occurrence in the one patient who should have been spared. It is easily understandable, therefore, that the ophthalmologist is hesitant in operating on a patient with syphilis, especially if it is in the active stage. Nevertheless, recent case studies have shown that the large majority of patients with syphilis may be operated on with impunity from the point of view of wound healing. However, those tissues which have already shown gummatous change should be left alone, as extensive and destructive results might ensue. When the indications point toward an operation, no time should be lost in advising it when it is deemed an emergency procedure. However, the postoperative course should include the institution of antisymphilitic therapy. Too frequently the operation is deferred until the ocular condition has advanced to the point at which the chances of arresting the process are slight. In cases in which therapy is not urgent, treatment of the systemic infection is indicated and may be followed at some future date by the necessary operative procedure.

SUMMARY AND CONCLUSIONS

Instead of presenting a statistical study of a group of cases of ocular syphilis in this paper, we have described our experiences in the treatment and observation of the disease. The accepted modern forms of treatment are indicated. The ideal antisymphilitic drug is arsphenamine or one of its congenitors. It should be given in conjunction with either mercury or bismuth compounds, in combined continuous courses over a period of years. Iodides should be used in all of the various stages and clinical phases of the disease. In the usual case of syphilis complicated by diplopia, disturbance of the ocular muscles, iritis or keratitis, especially

before degenerative changes have set in, the routine methods of anti-syphilitic therapy are sufficient to prevent and cure the invasion of the optic structures. Herxheimer reactions are usually avoided if two or three preliminary injections of a mercury or a bismuth compound are given before the institution of arsphenamine. If a Herxheimer reaction develops, treatment should not be stopped if destruction of tissues is to be prevented. Neurorecurrences are usually the result of inadequate or short courses of intermittent treatment.

Arsphenamine and neoarsphenamine may be replaced by mapharsen, silver arsphenamine or sulfarsphenamine as already indicated. In cases in which there is no response to routine treatment, and especially when there are progressive changes, injections of tryparsamide together with the administration of bismuth compounds or iodides should be given for at least a year. Precautions should be taken against atrophy of the optic nerve.

The Swift-Ellis and the Swift-Ellis-Ogilvie method of intraspinal therapy should be tried in cases of resistant ocular syphilis associated with neurosyphilis. Such treatment will often reverse the spinal fluid reaction to normal and occasionally improve the clinical picture.

The other form of supplemental treatment, hyperpyrexia, produced by inoculation with malaria, injections of biologic products or mechanical means, may reverse the state of the spinal fluid in over 40 per cent of cases and arrest the clinical progress in about 15 per cent. The patients who receive the least beneficial response from therapy are those who have had neurosyphilis for more than ten years and present old lesions of a degenerative type plus severe involvement of the spinal fluid. Reversals in the state of the blood and spinal fluid do not always indicate satisfactory clinical improvement.

The recent trends in the therapy of ocular syphilis offer a much more hopeful prognosis than is generally believed. The institution of the newer remedies together with those proved efficacious over a period of years will greatly lessen the incidence and prolongation of ocular complications.

2 East Fifty-Fourth Street.

IRITIS DUE TO BACTERIA AND BACTERIAL TOXINS ASSOCIATED WITH DENTAL SEPSIS

EXPERIMENTAL PRODUCTION IN LABORATORY ANIMALS BY
ADMINISTRATION OF THESE TOXINS

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The relation between ocular diseases and oral sepsis has long been recognized. Articles¹ on ocular diseases caused by dental infection have appeared in the dental literature as far back as 1839 and serve to establish the recognized clinical association between diseases of the mouth and diseases of the eye. The medical literature is replete with experimental investigations concerning the production of iritis by the administration of various bacterial agents obtained from multiple body foci, through various routes of administration. Most of the experimental workers hopefully allied themselves in their research with the attractive theory of Rosenow² (1915), viz., that bacteria have a specific tendency to localize in certain tissues of the body, dependent on some peculiar inherent property, and based their studies on this concept. However, this present work is more especially concerned with the particular role of dental sepsis in the production of iritis experimentally.

The fact that many observers who have made contributions to this subject used methods that have not been uniform, obtained results at variance one with the other and drew widely different conclusions seems to suggest that the subject is surrounded by much that is as yet unknown. It seemed desirable, therefore, to throw some further light on the investigative work accomplished thus far by presenting a series of laboratory experiments on albino rabbits, in which I studied the effects of the bacteria and bacterial toxins found in the diseased processes of infected human teeth on the production of iritis in these animals.

In attempting to elucidate further the possible etiologic role of dental sepsis in the production of iritis, one must take cognizance of the many difficulties to be encountered in a work of this kind. The acute

From the Department of Ophthalmology, St. Louis University School of Medicine.

1. Black, A. D.: Ocular Diseases Resulting from Dental Lesions, *Ophth. Rec.* **24**:610, 1915.

2. Rosenow, E. C.: Iritis and Other Ocular Lesions on Intravenous Injection of Streptococci, *J. Infect. Dis.* **17**:403, 1915.

inflammation caused by the injection of a moderately heavy suspension of bacteria locally may result in panophthalmitis with destruction of the eye. Some of the animals used in the present study acquired septicemia and died during the experimental observations. The bacteria themselves, it was found, are markedly sensitive to oxygen, so that growth during culture occurred only under conditions of reduced oxygen tension. In some instances, aerobic cultivation tends to destroy the characteristics of the organism for elective localization. Then, too, not all dental foci yield micro-organisms which have elective localizing power. Rosenow³ stated, in this regard, that not every streptococcus has a predilection for the eye. Moreover, he⁴ expressed the belief that a prolonged contact of bacteria with leukocytes in the pus and a high oxygen tension in the mucus on the surface of mucous membranes appear to reduce the invasive powers of the bacteria. The length of existence of the bacteria in the host must also be considered. In man it must be assumed that these bacteria were present in the mouth for months and even years, the iris thus being subject to their influence over a long period of stimulation. Finally, it must be remembered that the degree of virulence and the ability to invade tissues will vary under changed conditions of habitat. Other factors of importance that play a role, such as generalized or local lowered resistance of the host, severe or repeated injuries and sensitization of the tissues, are, of course, also to be considered.

Although rabbits are susceptible to the pathogenic action of many bacterial species, one might well argue that positive findings cannot be considered as conclusive evidence of a parallel relation to ophthalmic disease in man. However, Metchnikoff⁵ made use of rabbits in his monumental work dealing with phagocytosis, and rabbits are constantly being utilized in laboratories today for both the study and the investigation of various human diseases. Rabbits are susceptible to streptococci and pneumococci and certain of the Spirochaetales in a manner that would suggest a measure of relation between the rabbit and man so far as susceptibility to certain kinds of pathogenic bacteria may be concerned. Furthermore, in those reactions related to a state of hypersensitiveness (e. g., serum sickness) the rabbit displays a capacity for reaction somewhat comparable to that in man.

3. Rosenow, E. C.: Results of Experimental Studies on Focal Infection and Elective Localization, *M. Clin. North America* 5:573, 1921.

4. Rosenow, E. C.: The Pathogenesis of Focal Infection, *J. Am. Dent. A.* 5: 113, 1918.

5. Metchnikoff, cited by Garrison, F. H.: History of Medicine, ed. 3, Philadelphia, W. B. Saunders Company, 1924, p. 629.

REVIEW OF THE LITERATURE

Literature Relative to the Clinical Aspects of Iritis and Dental Sepsis.

—Among early observers who began to recognize the correlation between iritis and dental infection, Butler,⁵ a British ophthalmologist, attached much importance to the part played by infective foci as a cause of iritis. In 1911 this observer stated that iritis is frequently due to toxins absorbed from pus pockets around teeth, in pyorrhea, in sinus infections and from other foci, including those of chronic otorrhea. In his review of 100 cases, he found the iritis in 12 per cent to be due to oral, nasal or other sepsis in and about the mouth. Lang⁶ in 1913 found the ocular diseases (not specified as iritis) in 71 of 176 cases to be due to dental infections.

TABLE 1.—*Etiologic Factors in a Study of Two Hundred Cases of Iritis by Irons and Brown (1916 and 1923)*

Infections	Number of Cases of Iritis		Total Number of Cases
	Alone	Associated with Other Infections	
Undetermined.....	0	7	7
Syphilis.....	12	26	38
Gonococcus.....	8	2	10
Tuberculosis.....	8	0	8
Dental infection.....	12	15	27
Tonsillar infection.....	26	27	53
Sinus infection.....	1	3	4
Genitourinary infection (nonvenereal).....	6	0	6
Other infections.....	3	0	3
No infection.....	0	0	3
Combined infection.....	0	41	41
Total number of cases.....			200

Other pioneers in this field include Irons and Brown,⁷ who in 1916 observed a series of 100 cases of iritis in which they considered dental sepsis as a definite factor of importance. In 1923 they⁸ observed a second series of 100 cases in which they attached due importance to the role of dental infections. In table 1 are presented the results of their studies with respect to probable etiologic factors, so far as their data permitted.

Fuchs⁹ (1924), de Schweinitz⁹ (1924), Ball⁹ (1927) and Parsons⁹ (1929) gave proper importance to dental disease as foci of infections underlying the production of iritis.

Elschnig,⁶ in 1925, observing more closely than ever before the possibility of dental sepsis as an etiologic factor, found the iritis in 26

6. Cited by Gifford.¹⁰

7. Irons, E. E., and Brown, E. V. L.: Etiology of Iritis, J. A. M. A. **66**: 1840 (June 10) 1916.

8. Irons, E. E., and Brown, E. V. L.: Etiology of Iritis, J. A. M. A. **81**: 1770 (Nov. 24) 1923.

per cent of a series of his cases due to this cause. He expressed the belief that a toxemia is produced by the products of disintegrated bacteria and pus present in dental foci.

Among other observers, Bulson⁹ (1925) noted that the iritis in 32 per cent of his series of 100 cases was due to infected teeth, whereas Newton⁹ (1925), in a series of 75 cases of uveitis, found dental sepsis to be the causative agent in 14.6 per cent. Gifford¹⁰ in 1930 studied the various etiologic factors in a series of 118 cases of iritis; he attributed the infection in 12.7 per cent of these to defective teeth.

It is interesting to note at this point that while Elschmig⁹ (1925) believed that a toxemia is caused by the products of disintegrated bacteria and pus present in dental foci, de Schweinitz, in his classic report before the International Medical Congress in 1913, expressed the belief that the production of iritis by bacterial toxins alone had never been proved and that the bacteria themselves are present in the lesions.

That a rather definite correlation exists between iritis and dental sepsis would seem to follow when one considers the curative effects that have ensued after the removal of dental foci. The extraction of the infected tooth or teeth, in some instances with curettage of the alveolar socket for remnants of septic material, resulted in a steady improvement in the eye, with ultimate recovery and no recurrence of the iritis. It seems reasonable to infer that the infective foci were the cause of the inflammation in the eye. The reason why no improvement has seemed to follow the removal of a septic tooth or a number of such teeth in some instances may be that there was incomplete evacuation of all of the diseased tissue. Some observers¹¹ have called attention to this fact and have reported several cases of iritis in which, though there was no improvement after dental extraction, decided improvement occurred on curettage of the alveolar sockets containing such infective areas.

In some instances the extraction of the infectious foci may, as an immediate result, give rise to acute iritis in an eye that previously had been quiet. In a personal communication Luedde stated (1939) that he had repeatedly noted an apparent focal reaction in ocular tissues when infectious teeth were extracted. In 2 cases the reaction was particularly severe. In 1 of these cases the patient, a man aged 40, had been under treatment elsewhere for recurrent iritis over a period of two years, no

9. Newton, F. H.: Analysis of Series of Cases of Uveitis with Special Reference to Etiology, *Texas State J. Med.* **21**:315, 1925.

10. Gifford, S. R.: Review of the Literature on the Etiology of Acute Iritis, *Am. J. Ophth.* **14**:100, 1931.

11. Narreau, O. F.: Relation of Dental Infection to Eye Conditions, *Dent. Items Interest* **56**:419, 1934. Lowell, W. H.: Relation of Focal Infection to Diseases of the Eye, *Dent. Cosmos* **76**:350, 1934. de Schweinitz, G. E.: Dental Sepsis in Its Relation to Ocular Disorders, *ibid.* **62**:565, 1920.

search for foci of infection having been made during this time. Roentgenographic examination revealed the presence of two teeth with considerable periodontal infection. Extraction of these was followed by a severe ocular reaction, including diminution of vision, severe pain and clouding of the anterior chamber. After two days the reaction subsided, and the condition cleared up without further recurrence. In the second case, the patient, a woman, first came under observation in 1920, when 40 years of age, for treatment of retinitis pigmentosa. Examination confirmed the previous diagnosis of retinitis pigmentosa and revealed the presence of incipient cataract in both eyes. Thirteen years later, with the subsequent development of the cataractous lenses, iridectomy was carried out on one eye as a preliminary procedure to see how the ocular tissues would react. Mild traumatic iritis followed, but this cleared up spontaneously within a week. Two months later, in preparation for operation for the cataract, a roentgenographic examination of the teeth revealed the presence of periodontal infections in eleven. These were extracted at one sitting, immediately after which acute iritis with copious exudation into the anterior chamber occurred (in the eye that was operated on). There were severe pain and elevation of the intraocular tension for several days. This exudate later became organized into a dense grayish mass, filling almost entirely the width and depth of the chamber. Five years later, cataract extraction with removal of the organized exudate from the anterior chamber was attempted. Recovery followed without any such complications as had been precipitated by the multiple extractions several years before.

Literature Relative to Experimental Work Previously Reported.—

In 1914 Rosenow² pointed out that streptococci from persons with rheumatic arthritis and myositis injected intravenously into animals are prone to lodge in the capillaries of the iris, producing iritis and other ocular lesions. Following this, with a series of animal experiments he was able in 1915 to produce iritis or iridocyclitis in a few instances after the injection of certain laboratory strains of streptococci cultivated from small pus pockets in the tonsils of normal persons.

Irons, Brown and Nadler¹² in 1916 presented a classic work on experimentation with rabbits. Iritis developed in 5 of 8 rabbits into which were injected intravenously suspensions in salt solution (of subcultures on blood agar) of a hemolytic streptococcus isolated from a human tear sac. Cultures of hemolytic streptococci taken from the same tear sac six weeks later failed to produce iritis in a total of 22 rabbits, whether other organisms from a broth culture inoculated directly from

12. Irons, E. E.; Brown, E. V. L., and Nadler, W. H.: The Localization of Streptococci in the Eye: A Study of Experimental Iridocyclitis in the Rabbit, *J. Infect. Dis.* **18**:315, 1916.

the patient or organisms in pure culture after isolation on blood agar were used for the injections. This loss of virulence seems to indicate that the invasive power of an organism for a special tissue may change within a period of time during residence in the host.

Veach,¹³ in 1920, in many series of experiments with rabbits, adopted the procedure of introducing the organisms directly into the anterior chamber. With a fine hypodermic needle, a puncture was made at the corneoscleral margin, the needle going into the base of the iris and as close to its anterior surface as possible, and a saline suspension of the bacteria obtained from patients with septicemia and otitis media and from the pulp of extracted decayed teeth was injected. The organisms used were *Staphylococcus aureus*, *Streptococcus haemolyticus* and *Streptococcus viridans*. In all six series of his report, moderate to severe iritis promptly occurred within twenty-four hours after injection.

Benedict¹⁴ (1920) reported a case in which the patient came under his care because of severe iritis recurring every spring and fall for over a period of six years. These attacks were always preceded by soreness of the right upper bicuspid, which developed about three days before the eye became inflamed. Frequently these attacks were also accompanied by general rheumatic pains, particularly if the patient had been exposed to bad weather. A general physical examination did not reveal any cause for the iritis, except dental infection. The tooth which the patient pointed out as bothersome was extracted, and a culture was made from its pulp. This culture was injected into a rabbit intravenously, after which iritis developed in both eyes within several hours.

Lewis¹⁵ (1923) had a patient with severe iritis who suffered also from an apical abscess; he advised that the offending tooth be removed and cultured in bouillon. The predominating organism found was a "*Streptococcus haemorrhagica*." He injected massive doses (6 cc.) of this culture intravenously into several white rabbits, and twenty-four hours later he repeated the injections intra-abdominally. He observed a definite iritis (bilateral) within forty-eight hours after the first injection.

Haden¹⁶ (1923) produced iritis and iridocyclitis in rabbits by the intravenous injection of streptococci and staphylococci isolated from chronic dental foci of patients suffering from ocular infections concomitantly and from healthy patients not suffering with concomitant ocular conditions. His observations illustrated, too, that a much higher

13. Veach, O. L.: Experimental Production of Iritis and Its Treatment with Foreign Protein, *Am. J. Ophth.* **3**:93, 1920.

14. Benedict, W. L.: Value of Dental Examination in the Treatment of Ocular Disorders, *Am. J. Ophth.* **3**:860, 1920.

15. Lewis, F. P., in discussion on Irons and Brown,^s p. 1775.

16. Haden, R. L.: Elective Localization in the Eye of Bacteria from Infected Teeth, *Arch. Int. Med.* **32**:828 (Dec.) 1923.

percentage of ocular lesions occurred in animals inoculated with bacteria which were isolated from those patients suffering from ocular diseases at the time. His case reports showed also that in many instances the iritis and uveitis improved definitely after the removal of the infected teeth.

Brown and Dummer¹⁷ in 1929 worked out a series of experiments in which their technic of administering the organisms showed a distinct line of departure from that of their predecessors. Desiring to establish a more satisfactory method of producing experimental iritis, they discarded the methods used by the aforementioned investigators. They believed that direct injection of the organisms into the eye created too great a source of error to render this method reliable and that the possibilities resulting from trauma and secondary infections were uncontrollable. They thought of intravenous and intraperitoneal injections as too haphazard, believing that secondary foci might be initiated which would change the characteristics of the organism or cause a generalized infection that would kill the animal before the desired localization took effect. They adopted the direct arterial route, dissecting the neck of the animal to free the carotid artery from its adnexae and injecting about 1 cc. of a heavy suspension of organisms directly into this vessel. Iritis was produced in twenty-four hours in 2 of 3 rabbits into which a strain of hemolytic streptococcus from the nasal secretions of a patient suffering from acute iritis was injected.

Brown¹⁸ (1932) claimed to have produced an acute inflammation of the uveal tract by the intraocular injection of a streptococcus toxin associated with scarlet fever. He did not, however, experiment with any toxins obtained from bacteria associated with dental infections.

Berens, Nilson and Chapman¹⁹ in 1936 produced iritis in rabbits by the intravenous injection of either primary or purified cultures obtained from various foci, such as the nose and throat, teeth and tonsils of patients with acute or chronic ocular inflammations, arthritis and thyrotoxicosis and, in some instances, of healthy subjects without demonstrable lesions. The organisms identified in the pure cultures consisted of staphylococci (*Staph. albus* and *aureus*), streptococci (alpha, beta and gamma types), enterococci, colon bacilli, nonlactose fermenters and Friedländer bacilli.

17. Brown, A. L., and Dummer, C.: The Experimental Production of Iritis, *Arch. Ophth.* **2**:573 (Nov.) 1929.

18. Brown, A. L.: Considerations Underlying the Experimental Production of Iritis, *Am. J. Ophth.* **15**:19, 1932.

19. Berens, C.; Nilson, E. L., and Chapman, G. H.: Iritis Produced in Rabbits' Eyes by Intravenous Injection of Bacteria Isolated from Patients with Certain Inflammatory Eye Diseases, *Am. J. Ophth.* **19**:1060, 1936.

One must not lose sight of the fact that while some research has been done on the question of dental foci and iritis, most investigators have concerned themselves only with the micro-organisms present in the dental lesions and few have considered the investigation of the effects of bacterial toxins produced in and about such dental foci.

PROCEDURE OF EXPERIMENTS

In order to ascertain the effects of bacteria and the bacterial toxins which are associated with dental infections on the iris of laboratory animals, the following experiments were performed. They were divided into five series, three concerned with the effects of toxins alone and the others with the organisms themselves. A general scheme of the entire experimental work is presented in table 2, which, it is hoped, will serve as a guide from time to time throughout the work.

TABLE 2.—*General Scheme of Experiments*

Series of Experiment	Material Used in Administration	Presence or Absence of Concomitant Iritis in Patients from Whom Teeth Were Obtained
1.....	Bacterial toxin A	Without iritis
2.....	Bacterial toxin B	Without iritis
3.....	Bacterial toxin C	With iritis
4.....	Bacterial organisms (suspension A)	Without iritis
5.....	Bacterial organisms (suspension B)	With iritis

The chief interest has centered, however, on whether or not the toxins alone were responsible for the ocular manifestations rather than the micro-organisms proper. Many previous investigators who have produced iritis in laboratory animals made use of virulent organisms obtained from various body foci. I wished to see, aside from this, what effect particularly the bacterial toxins from dental foci alone would have on the eye.

SERIES 1.—*Injections with Bacterial Toxin A.*—Twenty-four teeth were obtained in the dental clinic under aseptic precautions immediately after their extraction from patients who had no demonstrable ocular lesions at the time. The condition of the teeth obtained throughout these experiments (both from clinical and from roentgenographic evidence) was as follows: Several had rarefied areas around their apices: several had been loose in their sockets and contained suppurative processes at the gum margins; several contained loose bulbous granulomas around their apices; some were abscessed, and a few had infected root canals; in a few instances retained roots which had been left in the lower cuspid and bicuspid regions were obtained.

With a sterile rongeur forceps, the tips of the apices of these teeth were cut from the main root stem (including the root canals however) and dropped into a small Erlenmeyer flask containing 25 cc. of sterile beef infusion broth. Beef broth was used in the beginning because the organisms present did not seem to

require especially nutritive mediums. After its contents were shaken vigorously for several minutes, the flask was placed in the incubator for three to four days to allow for bacterial growth and activity and for the production of exotoxins. Meanwhile, cultures of this suspension on blood agar plates revealed the following organisms to be present: nonhemolytic streptococci; nonhemolytic staphylococci; nonhemolytic hemophili; hemolytic hemophili, and *Str. viridans*.

At the end of the period of incubation, the entire suspension, containing apexes, granulomatous material, spicules and material curetted from the root surfaces, was filtered clear and the filtrate placed over a water bath at 60 C. for one hour in order to kill the organisms present. Subsequent subcultures from this proved that no live organisms were present.

TABLE 3.—*Results Obtained with Toxin A*

Method of Administration	Ocular Involvement	Other Forms of Involvement
Subconjunctival injections in rabbits 1, 2, 3 and 4 with increasing doses of 2 to 8 minims, respectively	No signs of iritis; suppurative conjunctivitis in rabbits 3 and 4	None
Subconjunctival injections in rabbits 1, 2, 3 and 4 with uniform doses of 1 cc. every 48 hours	No signs of iritis; suppurative conjunctivitis in rabbits 1 and 2	None
Subcutaneous injections in rabbits 2 and 4 of 2 cc. every 48 hours	None	None
Intradermal injections in rabbits 1, 3, 5 and 6 of 0.5 cc. into the skin over the neck	None	Mild local erythema of the skin persisting 18 to 36 hours
Intravenous injections in rabbits 1, 3, 5 and 6 of doses of 1.5 cc.; injections repeated	None	Rabbit 5 became quite ill for 3 to 4 days
Instillations into the conjunctival sac alone in rabbits 1, 3 and 5	None	None
Instillations into the conjunctival sac combined with physostigmine salicylate in rabbits 2, 4 and 6	None (marked pupillary contraction)	Systemic reactions of marked tremors of the body and leg, due to poisoning from physostigmine salicylate
Instillations into the conjunctival sac with injury to the iris in rabbits 1, 3 and 5	No signs of iritis; slight ciliary redness over the site of the needle puncture	None

The resultant suspension was designated as toxin A for purposes of identification and investigated for its experimental effects.

Four healthy albino rabbits, weighing approximately 2,000 Gm. and from stock previously unaffected by immunization or sensitization experiments, were given a subconjunctival injection of toxin A in the right eye, the dosage varying from 2 minims (0.12 cc.) for the first rabbit to 8 minims (0.49 cc.) for the fourth, with increases of 2 minims for each succeeding animal. Before each injection, the conjunctival sacs were instilled several times with a 1 per cent solution of pontocaine hydrochloride and the sac flushed out with saline solution. The left eye of each rabbit was used as a control.

Observations were made in four, eight, twelve, twenty-four and forty-eight hour periods in each case. At the end of the twenty-four hour period, a mild to moderately severe suppurative conjunctivitis developed in rabbits 3 and 4, which cleared up spontaneously in two or three days. No signs of iritis appeared at any time throughout these periods (table 3).

The foregoing procedure was repeated for the same 4 rabbits after one week's rest, an increased amount (1 cc.) being used for the injections, which were repeated every forty-eight hours for three administrations. As in the previous experiment, no signs of iritis developed at any time, although a mild conjunctivitis developed in rabbits 1 and 2, which spontaneously cleared up within a few days. At no time were any of the animals observed to be ill.

To test the potency of the toxin suspension, rabbits 1 and 3 of this series and 2 others from a fresh stock were given intradermal injections of 0.5 cc. of the toxin into a shaved area on the back of the neck. A mild local cutaneous reaction, consisting of a moderately large circular erythematous area (about the size of a 5 cent piece), developed in all 4 rabbits; it resolved within two days. Similar injections made with sterile beef broth infusion in the same amounts failed to elicit a similar cutaneous reaction.

To ascertain whether or not the subcutaneous route of administration could be effective, rabbits 2 and 4 were given subcutaneous injections of 2 cc. of the toxin every two days for several administrations. No signs of iritis appeared (table 3).

It was then decided to administer the toxin suspension intravenously, using 2 of the original rabbits (nos. 1 and 3) and 2 from a fresh stock (nos. 5 and 6). Animals 1 and 5 each received 1 cc. intravenously in the vein of the right ear, and animals 3 and 6 each received 1.5 cc. in the vein of the right ear. At no time did any signs of iritis develop, though rabbit 5 became ill at the end of twenty-four hours, but recovered. Repetition of the injections in the same amounts after one week's rest did not produce any positive results.

It was next decided to try the effects of the toxin A on the iris by direct instillation into the conjunctival sac: first, alone; secondly, in conjunction with the instillation of physostigmine salicylate, and thirdly, in conjunction with direct irritation (trauma) to the iris by means of a sterile needle. The reason for using the physostigmine salicylate with the toxin was to observe if the combined effects of the two would more readily produce iritis. The strong contraction of the pupil, it was thought, during the presence of the toxin might well serve to initiate the inflammatory activity of the iris.

All 6 rabbits in this group were used throughout these procedures. The right eyes were used for the administration of the toxin and physostigmine salicylate, the left eyes being left as controls. The toxin was instilled every day for a ten day period in rabbits 1, 3 and 5, while rabbits 2, 4 and 6 received both instillations of toxin and drops of 1 per cent aqueous solution of physostigmine salicylate. About 3 to 5 drops of toxin and 1 to 2 drops of physostigmine salicylate from the end of a fine dropper were used. Observations were made two or three times each day during the course of this experiment, and at no time did any signs of iritis appear in any of the 6 animals. On the first day rabbits 2, 4 and 6 suffered from the effects of poisoning with the physostigmine salicylate, due obviously to an overdose. The original dose (3 to 5 drops) was reduced thereafter, and no further disturbing effects from this drug were noted (table 3).

After studying the combined effects of toxin and physostigmine salicylate, it was decided to investigate the combined effects of toxin and direct irritation of the iris. After a week's rest, the same 6 animals were used, as follows: Rabbits 2, 4 and 6 received daily instillations in the right eyes of 2 to 5 drops of toxin A suspension, while rabbits 1, 3 and 5 likewise received daily instillations of toxin in the right eyes plus direct irritation of the iris of the same eyes every other day. The latter procedure was accomplished as follows: Four to 6 drops of a 1 per cent solution of pontocaine hydrochloride was dropped over the cornea into the sac, followed a few minutes later with a copious flush with saline solution through the entire sac. The tip of a small sterile needle, fixed into a wooden handle, was

admitted through the upper and outer limbus directly into the anterior chamber and in front of the plane of the iris; its point was then drawn over the base of the iris to and fro for a few strokes and quickly withdrawn. A small spot of ciliary redness appeared at the site of entry of the needle shortly after and remained for several hours, but later disappeared. The trauma to the iris was repeated in the 3 animals mentioned every other day for four times, but except for the small amount of ciliary redness appearing only at the site of puncture, which was considered merely the immediate result of the needle passing through tissue, no definite signs of iritis appeared at any time throughout this series. The pupil remained always well reactive, equal in size to its fellow and regular in shape. The iris itself appeared normal when compared with its fellow. At no time did the faint redness appearing in scattered areas of the surrounding conjunctiva assume the dignity of a true circumcorneal injection (table 3).

TABLE 4.—*Results Obtained with Toxin B*

Method of Administration	Ocular Involvement	Other Forms of Involvement
Subconjunctival injections in rabbits 1 and 2 of 1 cc. of toxin	No signs of iritis; suppurative conjunctivitis in rabbits 1 and 2	None
Subconjunctival injections in rabbits 1 and 2, after 1 week rest, of 1 cc. of toxin every 48 hours	None	None
Intradermal injections of 0.5 cc. in rabbits 1 and 3	None	Local erythema of the skin 28 to 48 hours
Subcutaneous injections in rabbits 1, 2 and 3 of 2 cc. every 48 hours	None	None
Intravenous injections in rabbits 1, 2 and 4 of 1 cc. of toxin; repeated after 48 hours in the same animals with 2 cc.	None	Rabbit 4 became severely ill after 2d dose and died 5 days later
Instillations into the conjunctival sac alone in rabbits 1, 2, 3 and 4	None	None
Instillations into the conjunctival sac in combination with physostigmine salicylate in rabbits 2 and 4	None (marked pupillary contraction)	None
Instillations into the conjunctival sac with injury to the iris in rabbits 2 and 4	No signs of iritis; slight ciliary redness over the site of the needle puncture	None

SERIES 2.—*Injections with Bacterial Toxin B.*—Since all negative results were obtained with the toxin made for the first series of experiments, it was decided to prepare a new supply from other dental material, using a different culture medium and method of extracting the toxin, which was designated as toxin B for identification.

Twenty teeth were again obtained in the dental clinics from patients who at the time manifested no ocular inflammation. Under aseptic precautions, these were prepared in a similar manner as before, except that 40 cc. of brain-heart infusion broth containing 0.1 per cent agar was used for the medium. This was considered more suitable as a medium for organisms that might not only require especially nutritive material for growth and activity but also be micro-aerophilic in their oxygen requirements.

Into each of two sterile test tubes, approximately 20 cc. (in order to give depth to the culture bed) of the freshly made suspension was poured and incubated for three to four days. Cultures made during this time showed the presence of the following organisms: hemolytic streptococci; *Str. viridans*; non-

hemolytic streptococci; hemolytic hemophili (pleomorphic gram-negative rods) and nonhemolytic hemophili; nonhemolytic staphylococci, and gram-positive spore bearers (aerobic).

The bacteria in one of these test tubes was then subjected to the water bath at 60 C. for one hour. Cultures of this subsequently proved sterile. The contents of the second test tube were passed through a Berkefeld filter, and cultures of the filtrate later proved also to be sterile. The resultant suspension and filtrate were then added to each other, mixed and labeled toxin B for further identification.

Four healthy albino rabbits selected from a stock previously unaffected by immunization experiments were used in this series. The results obtained are tabulated for clarity and convenience in table 4.

In order to avoid the repetitious use of terms and the voluminous details of procedure in the administration of the toxins and suspensions of bacteria and to present the experimental data in as clear and understandable a manner as possible, it was deemed advisable to make tabulations of all results obtained at the end of each new experimental series (similar to the manner shown in table 3). The amounts used in the injections were practically identical with those used previously. All details and steps in administration were the same. Right eyes were always used for the local injections and the left eyes for controls.

SERIES 3.—*Injections with Bacterial Toxin C.*—Having obtained negative results thus far with bacterial toxins associated with septic dental processes from patients unaffected at the time with ocular inflammation, I then proceeded to investigate the effects of toxin which was obtained from dental foci in patients who did manifest uveal inflammation at the same time.

The first patient, a woman aged 48, came to the clinic with a markedly acute iritis of the left eye and a large corneal ulcer; the onset occurred without an apparent external cause and was followed one week later by involvement of the other eye. General work-up in the various departmental clinics and laboratory tests gave essentially negative results, except for four upper incisors which were extremely loose in their sockets and showed greenish suppurative processes around the gum margins. Roentgenograms showed the presence of rarefied areas in the region of two apexes. Extraction of all four offending teeth was followed in one week by marked improvement in both eyes, with total disappearance of symptoms in the original eye.

The second patient, a man aged 36, previously in excellent health, had acute iritis in the right eye which had developed without apparent cause. Clinical and laboratory examinations gave essentially negative results. External examination of his teeth showed the presence of one upper cuspid to be extensively involved with ulceration. A roentgenogram indicated one small area of bone absorption near the apex. Removal of this tooth after one week's unsuccessful treatment with the usual methods resulted in prompt recovery.

The third patient, a man of about 69, came to the ophthalmic clinic with acute iridocyclitis of the right eye, with pus in the anterior chamber and ulceration of the cornea. Rigid treatment included, among other measures, the removal of several teeth, various states of caries, abscesses, retained roots and suppurative processes at the gum margins being present. Examination revealed the presence of an infection of an antrum, chronic prostatitis and hypertension. Improvement followed dental treatment, and the clearing up of other foci was rather protracted. Eventually, after several months of rigid care, the corneal ulcer healed and the ocular inflammation subsided.

From the teeth extracted from these three persons a toxin (C) was prepared in the manner similar to that worked out in the second series, viz., by using brain-

heart infusion broth with 0.1 per cent agar and extracting the toxin by passing the three to four day old bacterial suspension through a Berkefeld filter. Individual preparations of toxin were made, of course, in each instance as the patient came to the clinic for treatment; but since all methods of preparation and subsequent administration of the toxins were repeated in identical fashion, I have, for purposes of brevity and clarity, combined the data into one composite group (table 5).

Bacterial cultures made at the time of this group showed the following organisms predominating: gram-positive spore bearers (aerobic); nonhemolytic staphylococci; nonhemolytic streptococci; pleomorphic gram-negative rods (hemolytic hemophili); hemolytic streptococci and alpha hemolytic streptococci (*Str. viridans*).

TABLE 5.—*Results Obtained with Toxin C*

Method of Administration	Ocular Involvement	Other Forms of Involvement
Subconjunctival injections in rabbits 1 and 2 of 1 cc. of toxin	No signs of iritis; mild suppurative conjunctivitis in rabbits 1 and 2	None
Subconjunctival injections in rabbits 1 and 2, after 1 week's rest of 1 cc. of toxin every 48 hours	None	None
Intradermal injections of 0.5 cc. in rabbits 1 and 3	None	Local erythema of the skin persisting 2 days
Subcutaneous injections in rabbits 1, 2 and 3 of 2 cc. every 48 hours	None	None
Intravenous injections in rabbits 1, 2 and 4 of 1 cc. of toxin; repeated after 48 hours in the same animals with 2 cc.	None	Rabbit 4 became somewhat ill but recovered
Instillations into the conjunctival sac alone in rabbits 1, 2, 3 and 4	None	None
Instillations into the conjunctival sac in conjunction with physostigmine salicylate in rabbits 2 and 4	None (marked pupillary contraction)	None
Instillations into the conjunctival sac with injury to the iris in rabbits 2 and 4	No signs of iritis; slight ciliary redness over the site of the needle puncture	None

Four healthy albino rabbits selected from a stock previously unaffected by immunization experiments were used in this series of experiments with toxin C. Amounts and methods of administration were the same as in series 2, the right eyes being used for instillations and injections and the left eyes for controls. The results are tabulated in table 5.

SERIES 4 AND 5.—Injections with Bacterial Suspensions A and B.—I wish next to confirm some of the positive results obtained by previous investigators in the production of experimental iritis in animals with bacterial organisms. Of the various routes of administration that presented themselves for consideration, I decided on two as the most desirable, viz: (1) direct intraocular implantation and (2) intravenous. For the intraocular technic, I followed the method of Veach,¹³ who made injections into the limbus with a fine hypodermic needle, going into the base of the iris and as close to its anterior surface as possible. This procedure was preceded with local anesthesia followed with a copious lavage of the conjunctival sac with saline solution, the latter being repeated after the injection was completed. For the intravenous injections, moderate (1.5 cc.) to heavy (3 cc.) doses of the bacterial suspensions were used.

The organisms used were obtained from twenty-four hour subcultures of the primary growth made during the second and third series of experiments. These consisted of two main groups: those obtained from dental foci in patients not

TABLE 6.—*Results with Injections of Bacterial Suspension A (From Foci of Patients Without Concomitant Iritis)*

Method of Administration	Ocular Involvement	Other Forms of Involvement
Intraocular injections of 0.2 cc. of suspension of organisms at the base of the iris in rabbits 1 and 1x*	Marked iritis appearing within 8 hours and persisting for several days; resolution on the fifth day	None
Intravenous injections in rabbits 2 and 2x of 1.5 cc. of suspension of organisms A	None	None
Intravenous injections in rabbits 1 and 2 of 3 cc. dosage (increased)	Moderate iritis appearing within 12 hours in both rabbits and disappearing on the second day	None
Intravenous injections in rabbits 1x and 2x of 3 cc. dosage (increased)	Moderately severe iritis appearing within 8 hours in both animals, in 2x greater than in 1x; resolution within 2 days	None

* Numerals without x signify freshly selected animals not previously given injections of proteins in these experiments. Numerals with x affixed thereto signify animals previously given injections of proteins in these experiments.

TABLE 7.—*Results with Injections of Bacterial Suspension B (From Foci of Patients with Concomitant Iritis)*

Method of Administration	Ocular Involvement	Other Forms of Involvement
Intraocular injections of 0.2 cc. of suspension of organisms at the base of the iris in rabbits 3 and 3x	Marked iritis in both rabbits within 6 hours, persisting for several days and resulting in panophthalmitis	None
Intravenous injections in rabbits 4 and 4x of 1.5 cc. of suspension of organism B	Ciliary redness in both rabbits in 8 to 12 hours with resolution at the end of 24 hours	None
Intravenous injections in rabbits 3 and 4 of 3 cc. dosage (increased)	Mild to moderately severe iritis in both rabbits after 8 hours with resolution in rabbit 3 after the second day; infection persisted in rabbit 4 until the third day	Both rabbits became slightly ill within 12 hours; but recovered
Intravenous injections in rabbits 3x and 4x of 3 cc. dosage (increased)	Mild iritis in rabbit 4x after 12 hours; disappeared in 2 days; marked iritis in rabbit 3x within 12 hours, persisting for 2 to 3 days	Rabbit 3x became ill after first day; grew worse and died on sixth day

suffering from demonstrable uveal inflammations at the time and those obtained from dental foci in patients with concomitant iritis. For purposes of identification, the former was called bacterial suspension A and the latter bacterial suspension B. The bacterial contents of group A corresponded in identity to those used in the second series of toxin B experiments, while the bacterial contents of group B corresponded in identity to those used in the third series of experiments with

toxin C. A review of the general scheme of experiments (table 2) at this juncture will illustrate this.

For these last two series, a total of 8 animals was used: 4 healthy albino rabbits (nos. 1, 2, 3 and 4) selected from a stock previously unaffected by immunization or sensitization experiments and 4 healthy rabbits (nos. 1x, 2x, 3x and 4x) selected from those used in the earlier experiments and subjected to injections of toxin intravenously. The reason for using the latter was to see what effect, if any, the injection of organisms would produce in animals previously subjected to injections of protein.

The results in both these final series of experiments are tabulated in tables 6 and 7, respectively. All positive results were checked by repeating the procedures involved with sterile brain-heart infusion broth (without organisms or toxin) and obtaining negative results therefrom.

SUMMARY AND CONCLUSIONS

Iritis failed to develop in albino rabbits subjected by various routes of administration to the effects of bacterial toxins associated with septic dental foci in man.

Bacterial toxins obtained from septic dental foci of patients either with or without concomitant iritis did not cause iritis experimentally in the rabbit.

The route of administration of the bacterial toxins did not alter their effects, except to produce general illness and death in a few rabbits when given in large doses by the intravenous method.

On the other hand, the administration of bacterial organisms associated with septic dental foci in man produced iritis in albino rabbits.

It seems possible that the bacterial strains obtained from patients who had concomitant iritis possess greater virulence than those obtained from patients without concomitant inflammation of the iris.

In rabbits previously given injections of bacterial toxin iritis subsequently developed when bacteria was injected.

It is evident that the cause of the inflammation of the iris in these animals is directly due to a bacterial attack on the tissue. The inflammation could not be reproduced by administration of bacterial products such as the toxins made in this experiment.

These experiments do not refute the concept that in man the production of iritis may be brought about as follows: Toxins diffusing into the blood stream from an active septic focus or foci in the body might sensitize the iris tissue during a period of months or years, so that subsequently when bacterial emboli, discharged into the blood stream from this same (primary) focus, chance to lodge in the capillaries of the iris they (emboli) find a conditioned field, enabling them to manifest their pathogenic potentialities.

Miss Helen K. Moran, of the department of bacteriology of the Firmin Desloge Hospital, assisted in the preparation of the bacteriologic material for these experiments.

ACCEPTANCE OF WEAK CYLINDERS AT PARADOXIC AXES

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I have often noted, and no doubt a similar observation lies within the experience of every oculist, that after determining at retinoscopic examination that the patient needs a weak cylinder with its axis horizontal, the patient decidedly prefers the cylinder at an angle exactly opposite; however, with the addition of more spherical correction, he promptly reverses his choice to the correct meridian. This reversal phenomenon does not occur when the true axis is in the vertical or oblique meridians.

I do not refer to cases of astigmatism in which a weak plus cylinder becomes converted into an equivalent minus cylinder at the opposite axis as the result of an accommodative effort on the part of the ciliary muscle. I refer to instances in which cyclopegia is well effected by homatropine or atropine and in which the question of accommodation does not enter and the sign of the cylinder remains unchanged.

The explanation of this phenomenon lies in the following partially known facts: The average test letter is more readily recognizable when the vertical components are clear and the horizontal components blurred than when there is a lesser but uniform blur of all the elements. The letter is least recognizable when the horizontal components are clear and the vertical components blurred.

It must be remembered that in the eye the vertical curvature of the cornea-lens system is responsible for the clarity of the horizontal elements of the test letters, while the horizontal curvature influences the proper focusing of the vertical components of the letters. Blurring of these components as they appear to the eye may be simulated by camera studies. The distortion produced by lenses before the focused camera is not exactly equal to the distortion of the retinal image that these same lenses would produce if placed before an atropinized emmetropic eye, but the differences are sufficiently small to permit the analogy. Placing a cylinder at axis 90 degrees before the eye changes the effective horizontal curvature.

From the Knapp Memorial Eye Hospital.

Figure 1 *A* shows the effect obtained with an otherwise perfectly focused camera by placing in front of it a -0.50 D. cylinder, axis 180 degrees. This distorts the horizontal components of the letters because it decreases the vertical curvature of the lens system. The horizontal curvature remains in focus, giving clarity to the vertical components of the letters. This effect is comparable to that produced by a simple hyperopic astigmatism of $+0.50$ D., axis 180 degrees.

Figure 1 *B* shows a setup with a -0.25 D. sphere in front of the camera. It will be noted that the 20/20 line is slightly less legible than in figure 1 *A*, notably the letters A, R, and N. Holding the photograph at arm's length makes this more evident. This effect is comparable to that produced by a hyperopia of $+0.25$ D.

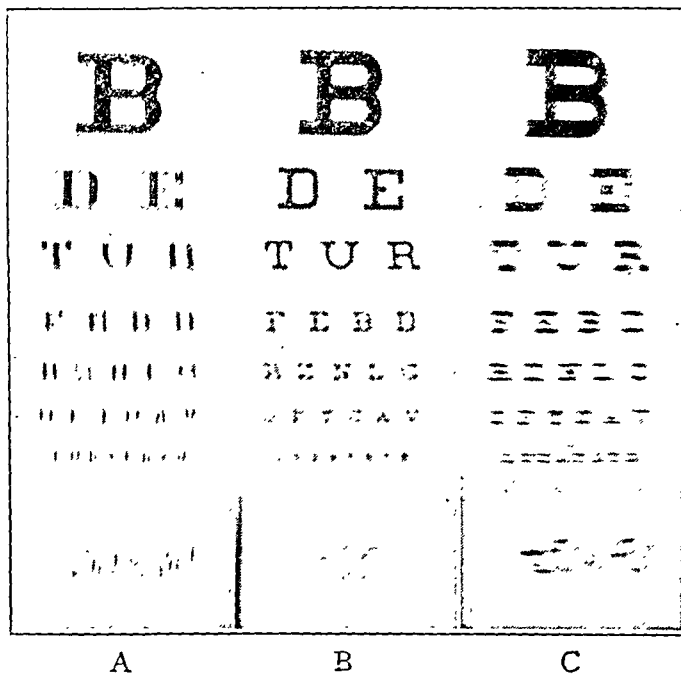


Fig. 1.—*A*, horizontal elements blurred 0.50 D.; vertical elements in focus. This effect is comparable to that produced by a refractive error of $+0.50$ D., axis 180 degrees. *B*, horizontal and vertical elements both blurred 0.25 D. This effect is comparable to that produced by a refractive error of $+0.25$ D. *C*, vertical elements blurred 0.50 D.; horizontal elements in focus. This effect is comparable to that produced by a refractive error of $+0.50$ D., axis 90 degrees.

Figure 1 *C* shows the effect of a -0.50 D. cylinder held at 90 degrees in front of the focused camera. The legibility is much less than in figure 1 *A* and *B*, because the vertical components are blurred. This effect is comparable to that produced by a simple hyperopic astigmatism of $+0.50$ D., axis 90 degrees.

Figure 2 shows diagrammatically the focal planes of the vertical and horizontal corneal curvatures in their relation to the retina, *R*, in a

case of hyperopic astigmatism (+ 1 D. sphere \subset + 0.25 D. cylinder, axis 180 degrees) when there is no lens in front of the eye (fig. 2 *A*); when the spherical correction is deficient by 0.25 D. and there is no cylindric correction (fig. 2 *B*); when the spherical correction is deficient by 0.25 D. and the cylinder is at the proper axis, 180 degrees (fig. 2 *C*), and when the spherical correction is deficient by 0.25 D. and the cylinder is at the improper axis, 90 degrees (fig. 2 *D*). Each space between the dots represents 0.25 D.

The effect of the correction in figure 2 *C* may be reproduced on the focused camera by a -0.25 D. sphere superimposed (fig. 1 *B*).

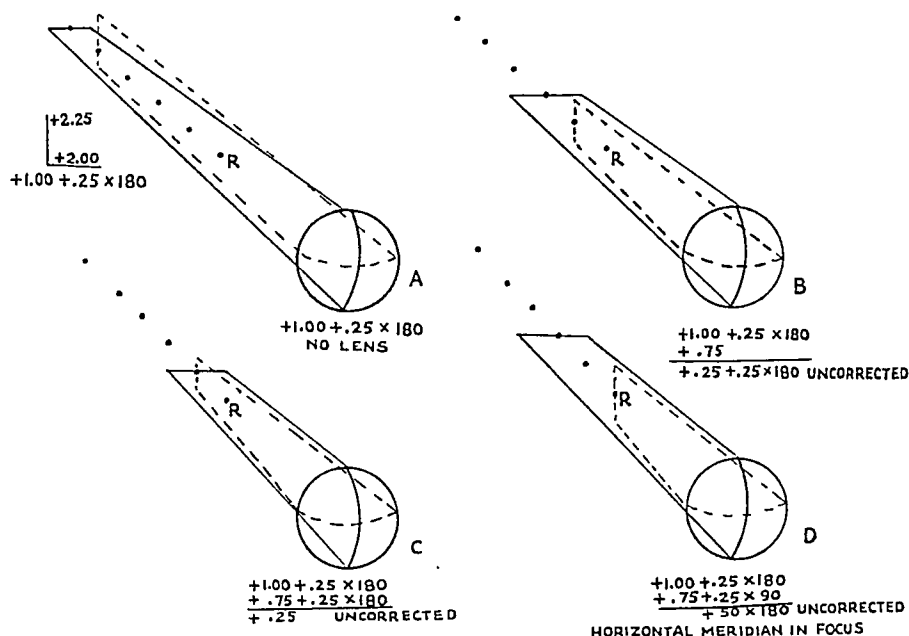


Fig. 2.—Diagrams representing the focal planes of the vertical and horizontal corneal curvature in their relation to the retina in a case of hyperopic astigmatism (+ 1 D. sphere \subset + 0.25 D. cylinder, axis 180 degrees) in various stages of correction. The partial correction in figure 2 *C* produces an effect comparable to that in figure 1 *B*. The partial correction in figure 2 *D* produces an effect comparable to that in figure 1 *A*. Given the choice between the correction in figure 2 *C* and that in figure 2 *D*, a patient may choose that in figure 2 *D*, wrong axis.

The effect of the correction in figure 2 *D* may be reproduced on the focused camera by a -0.50 D. cylinder, axis 180 degrees (fig. 1 *A*).

The patient in this particular case, having an undercorrected spherical error, may, when confronted with a choice of cylinder at the right axis (180 degrees, as in figure 2 *C*) and the wrong axis (90 degrees, as in figure 2 *D*), at times choose the wrong axis, because the vertical components of the letters are then in focus. With the addition of 0.25 D. more spherical correction, he will reverse his choice to 180 degrees.

Figure 3 shows diagrammatically the focal planes of the vertical and horizontal corneal curvatures in relation to the retina, *R*, in a case of hyperopic astigmatism ($+1$ D. sphere $\subset +0.25$ D. cylinder, axis 90 degrees) when there is no lens in front of the eye (fig. 3 *A*); when the spherical correction is deficient by 0.25 D. and there is no cylindric correction (fig. 3 *B*); when the spherical correction is deficient by 0.25 D. and the cylinder is at the proper axis, 90 degrees (fig. 3 *C*), and when the spherical correction is deficient by 0.25 D. and the cylinder is placed at the improper axis, 180 degrees (fig. 3 *D*).

The effect of the correction in figure 3 *C* may be represented by figure 1 *B*, and that in figure 3 *D*, by figure 1 *C*. The patient in this case,

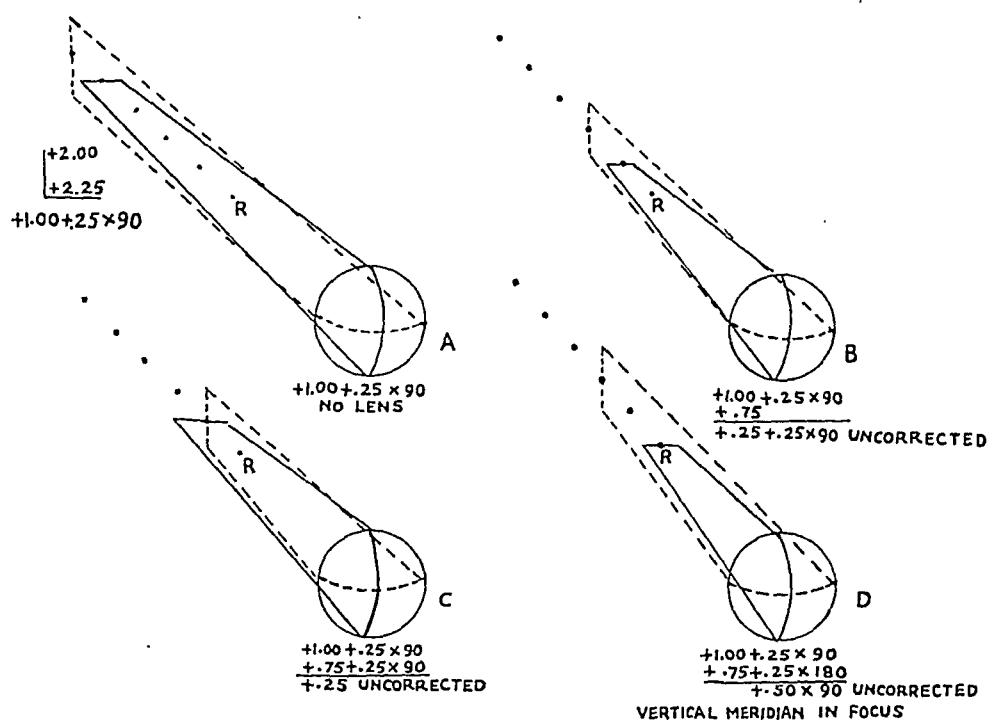


Fig. 3.—Diagrams representing the focal planes of the vertical and horizontal corneal curvatures in their relation to the retina in a case of hyperopic astigmatism ($+1$ D. sphere $\subset +0.25$ D. cylinder, axis 90 degrees) in various stages of correction. The partial correction in figure 3 *C* produces an effect comparable to that in figure 1 *B*. The partial correction in figure 3 *D* produces an effect comparable to that in figure 1 *C*. Given the choice between the correction in figure 3 *C* and that in figure 3 *D*, a patient will choose that in figure 3 *C*, right axis.

although having an undercorrected spherical error, will choose the proper axis (90 degrees), as in figure 3 *C*, and will reject the improper one (180 degrees), as in figure 3 *D*, because the vertical components of the letters are then out of focus. The addition of more spherical correction to make up for the deficiency will improve his visual acuity and will confirm the choice of axis 90 degrees.

A similar set of diagrams may be constructed for a myope with a weak cylinder at 180 degrees and at 90 degrees, except that the focal planes of the corneal curvatures will lie in front of the retina.

Suppose at refraction a myope requires a cylinder of -0.25 D., axis 180 degrees, and the spherical error is undercorrected by -0.25 D. If the cylinder is placed in the proper axis, the patient sees just as a person with uncorrected myopia of -0.25 D. sees. If the cylinder is turned to the improper axis of 90 degrees, the vertical corneal curvature is undercorrected by 0.50 D., but the horizontal curvature is made emmetropic, causing the upright components of the letters to stand out clearly. This is frequently more acceptable to the patient than the uniform blur present with myopia of -0.25 D., and he will demand the wrong axis. The addition of a -0.25 D. sphere will cause the patient to reverse his choice of cylinder to one at the proper axis of 180 degrees.

Suppose a myope requires a cylinder of -0.25 D. at axis 90 degrees. With the spherical error undercorrected by 0.25 D., placing the cylinder at the correct axis of 90 degrees will leave a spherical myopia of -0.25 D. Rotating the cylinder to 180 degrees will undercorrect the horizontal corneal curvature by 0.50 D. but will fully correct the vertical corneal curvature. The horizontal components of the letters will stand out clearly, while the upright parts will remain blurred. This is by no means as acceptable to the patient as the uniform blur of the myopia of -0.25 D. when the cylinder is placed at axis 90 degrees, and he will not demand the wrong axis. Adding the -0.25 D. sphere confirms the choice of axis at 90 degrees.

Both hyperopes and myopes, then, who on retinoscopic examination are shown to require a 0.25 D. cylinder, axis 180 degrees, may, and frequently do, choose the cylinder at axis 90 degrees if their spherical error is undercorrected by 0.25 D. If the deficiency in the spherical correction is added, the cylinder is promptly accepted at 180 degrees. In order for the cylinder when placed in the wrong axis to cause full correction of the horizontal corneal curvature, the sphere must be deficient by an amount equal to the strength of the cylinder.

If a hyperope or a myope requires a 0.25 D. cylinder, axis 90 degrees, as shown by retinoscopic examination, he is not likely to accept the cylinder at the wrong axis (180 degrees) even though the spherical error is undercorrected by 0.25 D.

This reversal phenomenon of the cylinder is met most frequently in myopes with an astigmatic error of 0.25 D. or 0.50 D. in the horizontal axis. It is apparent that in such cases the spherical error has to be fully corrected before reliance can be placed on the choice of axis.

A cylinder of 0.75 D. or more at axis 18 degrees is not likely to be accepted at a contradictory axis for two reasons. First, the refractionist

is not likely to undercorrect the spherical error by 0.75 D. or more; as I have noted previously, one of the conditions of the phenomenon calls for a deficiency in the amount of the sphere equal to the strength of the cylinder. If the sphere is deficient by an amount less than the strength of the cylinder, neither meridian will be in focus and the patient will not definitely accept either axis. Second, if the corrective cylinders were offered the patient while the spherical correction was deficient by 0.75 D. or more, the images would be so blurred for both the proper and the improper axes that he would accept neither axis very decisively.

When the cylinder lies at 45 or 135 degrees and the patient's spherical error is undercorrected, he will always accept the proper axis, because a 0.25 D. spherical deficiency will be preferred to the distortion produced by a 0.50 D. cylinder at 135 and 45 degrees. The closer the cylinder lies to 180 degrees the more likely is the occurrence of the reversal phenomenon.

One does not often encounter this phenomenon in hyperopes because one always starts refraction with a high plus sphere and works downward; by so doing one is not likely to undercorrect the spherical error. If undercorrection does occur, the phenomenon will be noted, unless, of course, the patient accommodates to make up for the lack of plus sphere, in which event he will, for the time being, have full spherical correction. Often some accommodative ability persists to mask the reversal phenomenon. It will, however, be noted with adequate cycloplegia.

For myopes, one proceeds from low to higher spheres and thus starts with an undercorrection. This, of course, tends to bring out the reversal phenomenon.

The myope who is wearing a slight spherical undercorrection will utilize the principles of letter recognition mentioned by looking at distant type through the sides of his lenses. He produces thereby a vertical astigmatic effect in the spheres and renders the horizontal corneal curvature more nearly emmetropic. This causes the legibility to be improved, because it clarifies the vertical moieties of the letters. With lenses of -2.00 to -4.00 D. of the old uncompensated type, this peripheral astigmatism may amount to 0.50 or 0.75 D. The subject will not peer through the upper or the lower edges of the lenses, as this will cause the induced cylinders to lie in axis 180 degrees, emphasizing the less desirable horizontal components.

COMMENT

Astigmats with a cylinder of 0.25 or 0.50 D. in the horizontal axis and a sphere which is deficient, respectively, by 0.25 or 0.50 D. will often accept the cylinder at the opposite axis (90 degrees). When

restitution of the deficiency in the spherical correction is made, the patient will demand the proper axis.

When the true axis lies at 90 degrees, this reversal phenomenon does not occur.

I occasionally utilize the reversal phenomenon to check for over-correction of sphere in myopic astigmatism, axis 180 degrees, when the cylinder is a weak one. If on reversing the sphere proportionately the cylinder is not accepted at the improper axis of 90 degrees, it is an indication that the spherical correction was too high originally.

The optical basis of this reversal phenomenon has been illustrated. It involves the comparative ease of recognition of test letters when the vertical or horizontal components are blurred as compared with a lesser but uniform blur of all the letter elements.

On this premise, simple astigmats have better visual acuity, as judged by the Snellen charts, when the cylinder lies at 180 degrees than when it is at 90 degrees. In other words, the visual acuity of astigmats is better when the horizontal corneal curvature is the emmetropic curvature. This explains the surprisingly good visual acuity of many persons with high astigmatism whose cylinder lies horizontally.

A person of early presbyopic age who has simple myopic astigmatism and who does not wear any correction is better off for reading if his astigmatism is at 90 than at 180 degrees. On the other hand, if the astigmatism is of the simple hyperopic type it should be easier for the patient wearing no lenses to distinguish near reading matter when the astigmatism is at 180 than at 90 degrees.

An anastigmat with an undercorrected spherical error is likely to have erroneously prescribed for him a weak cylinder, axis 90 degrees. By the same token, in an anastigmat, if the spherical error is fully corrected and a weak cylinder wrongly prescribed, it should be better tolerated at axis 180 degrees than at 90 degrees.

The practical application of this discussion may be embodied in the following rule: When a weak cylinder in the horizontal axis is expected to be correct from retinoscopic examination but is demanded by the patient in the contradictory vertical axis, add more spherical correction and the patient will reverse his choice to the proper axis.

In case of doubt while refracting at this stage, the elimination of test letters by substitution of an astigmatic cross will avoid psychologic problems of letter recognition and will avert discrepancies of axis.

Dr. Adolph Posner made the photographs necessary for this work.

TUMOR OF THE THIRD VENTRICLE WITH RESULTING CHRONIC INTERNAL HYDROCEPHALUS

CLINICAL HISTORY OVER A PERIOD OF SEVENTEEN YEARS

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PHILADELPHIA

Tumors of the third ventricle which cause chronic internal hydrocephalus are not uncommon, but the case here reported is interesting clinically because of the fact that the patient was followed for seventeen years and that death was due to a traumatic subdural hemorrhage.

The symptoms of a tumor of the third ventricle depend on the size and location of the growth and the involvement, either directly or through distant effects, of the important structures surrounding the ventricle. Weisenburg's¹ paper published in 1910, in which he reported typical cases, gave a good summary of these symptoms, as they were caused either by direct pressure or after extension into the aqueduct of Sylvius by growth of the tumor ventrally. They were as follows: paralysis of associated ocular movements and of convergence upward and, less commonly, to either side or downward; ataxia of the cerebellar type, as shown by the gait; stationary or voluntary movements of the limbs; occasional ptosis of the upper eyelids and protrusion of either one or both eyeballs, and, as a rule, large pupils with impaired reactions, paresis of the limbs of one or both sides and the general symptoms of tumor, such as headaches, choked disk, nausea, vomiting and vertigo. Tumor of the third ventricle does not cause specific mental symptoms; the occurrence of such symptoms is dependent on the compression of the cortex against the skull, resulting from internal hydrocephalus.

Fulton and Bailey² reported a series of cases of tumor in the region of the third ventricle from Cushing's service at Harvard Medical School in 1929 and gave a further important résumé of the symptoms produced by the growths, with an extensive bibliography. Other cases have

Read before the Philadelphia Neurological Society, April 28, 1939.

1. Weisenburg, T. H.: Tumours of the Third Ventricle with the Establishment of a Symptom-Complex, *Brain* **33**:236-260, 1910.

2. Fulton, J. F., and Bailey, P.: Tumors in the Region of the Third Ventricle: Their Diagnosis and Relation to Pathological Sleep, *J. Nerv. & Ment. Dis.* **69**: 1, 145 and 261, 1929.

been reported in the intervening ten years, but Dandy³ summarized the present knowledge as follows:

There is no syndrome of the third ventricle, *per se*, but tumors immediately affecting this cavity may cause characteristic symptoms by pressure upon its walls, and their contained nuclei and tracts. Among the well recognized syndromes due to lesions of these structures we may mention: (1) the infundibular syndrome (polyuria, adiposity); (2) the syndrome of the central gray matter around the posterior end of the third ventricle and aqueduct of Sylvius (hypersomnia); (3) the thalamic syndrome (central pain, painful hypesthesia); (4) the extrapyramidal syndrome (bradykinesia, rigidity); (5) the decerebrate syndrome (hypertonicity, Magnus-de Kleijn reflexes); (6) the syndrome of Parinaud (paralysis of conjugate vertical movements of the eyeballs); (7) the syndrome of the body of Luys (hemichorea; vide Martin, 1927, Eward, 1891); (8) the hypopituitary syndrome (infantilism, hypotrichosis, lowered metabolism); (9) the uncinate syndrome (olfactory and gustatory symptoms; vide Herzog, 1928); etc., etc.

All of these syndromes are of as definite localizing value in cases of brain tumor as any other symptoms when they occur precociously before the onset of pressure symptoms. The infundibular syndrome and the hypersomnia associated with lesions of the central gray matter are deserving of especial emphasis since, owing to imperfect understanding of their localizing value, they are seldom given the importance which they merit in localizing tumors of the brain.

REPORT OF A CASE

E. W. I., a 10 year old white boy, was first seen by me on Dec. 28, 1920. He had been sent from school because of defective vision, headaches, lack of concentration in school, nervousness and occasional attacks of nausea and vomiting. Vision of the right eye was 5/6 (partly) and of the left eye 5/9 (partly). After correction of a compound hyperopic astigmatism, this was improved to 5/6 in the right eye and 5/7.5 in the left. There were a vertical tip of one eye, amounting to 2 prism degrees, and decided hydrocephalus, the head measuring 61.5 cm. in circumference. The boy wore a hat with a 7 $\frac{3}{4}$ head size.

Examination of the eyegrounds showed bilateral low grade optic neuritis, with

surrounding retina. The child was at once referred to the family physician, Dr. Herman B. Allyn, who had examined him five years before. His notes made on May 22, 1916, when the child was 5 years old, recorded a large head and scars resulting from a forceps delivery. At the examination in 1920 Dr. Allyn found incoordination of arm and hand movements. The child was restless and uneasy and inclined to try to be funny and smart; lack of judgment was reported by his mother. He had a good memory and was good in school work, but he lacked the ability to concentrate.

He was referred to Dr. Charles H. Frazier at the University Hospital for study and roentgen examination of the skull. On Jan. 7, 1921, Dr. Frazier reported signs of increased intracranial pressure, as evidenced by the headache, vomiting and papilledema. In addition to this, he had signs of disturbed cerebellar function. There was marked ataxia in the movements of the upper extremities, together with dysmetria. In order to relieve the pressure and to prevent atrophy of the

3. Dandy, W. E., in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1932, vol. 12, p. 1.

optic nerves, Dr. Frazier advised subtemporal decompression and possibly a callosal puncture, although unfortunately the effects of the latter as a means of ventricular drainage were of comparatively short duration.

Suboccipital decompression was suggested as an alternative in the hope of relieving some of the cerebellar disturbances.

Consultation with Dr. de Schweinitz was asked, and on March 7 he confirmed the diagnosis of optic neuritis and beginning atrophy of the optic nerves. He also advised a decompression operation. Dr. Allyn agreed to this, and on April 14 Dr. Frazier did a suboccipital decompression. His notes made at the time of operation follow: "Bone was removed over both cerebellar hemispheres. The dural tension was much above normal but not extreme. There was a small collection of subdural fluid on the outer side of the left hemisphere, at which point the dura was punctured, and the fluid escaped as though under considerable pressure. After the introduction of a grooved director in the direction of the cisterna basalis, there was no escape of fluid." Dr. Frazier's conclusion was: "The absence of a large collection of the fluid in the posterior fossa and the evidently increased pressure lead one to the conclusion that the pressure must be due to internal hydrocephalus, with partial, but probably not complete, obstruction."

So far as the records show, a ventriculogram does not seem to have been made at this time.

After the operation the boy vomited frequently for three days, and there was an increase in the papilledema to 4 diopters and marked tortuosity of the retinal vessels. The neuritis slowly decreased, and as the patient's general condition had improved he was discharged from the hospital on May 21. Vision improved slowly, and the fields of vision gradually widened. Increased diplopia, however, appeared due to paresis of the right superior rectus muscle (oculomotor disturbance). Four months later the margins of the disks did not show any blurring, and the boy was able to return to school after the occipital wound healed. During the following three years he was seen frequently by me and by Drs. Frazier and Grant. Gradual deterioration of his mental condition became apparent. It became increasingly difficult to perform functional tests. The boy was impatient and resentful and at times refused to complete tests of the visual fields. In December 1924 he fell coming home from school, striking the left side of his head very hard. He complained of headache afterward and later had an attack of unconsciousness at home. There was no blurring of the nerve heads, and the fields had widened materially. The head measured 65 cm. in circumference at this time.

Several attacks of unconsciousness occurred, and in June 1925 he was readmitted to the University Hospital. The attacks were not accompanied by an aura. There were no convulsive movements, crying or other symptoms except for headaches, and they were of short duration. He was reported to be somewhat opinionated, stubborn and secretive but had kept up well in his school work. He was devoted to his mother but showed no sexual precocity or overdevelopment of the sexual organs. On June 19, 1 cc. of normal dye was introduced directly into the ventricle, a local anesthesia being used. The ventricular pressure measured 20 mm. Twenty minutes later a lumbar puncture was done, but none of the dye was recovered. Five and one-half hours after the operation urine was collected. It did not contain any dye; so there was every evidence that the ventricle, although not under great pressure, was obstructed. A roentgenogram of the skull showed the sella turcica to be slightly enlarged and the cerebral convolutions markedly atrophic. Vision in the right eye was reduced to 6/9 and in the left to 6/12.

There was optic neuritis, and vision measured at my office three months later was 5/7.5 (?) in the right eye and 5/12 in the left, the atrophy of the optic nerve being more marked in the left eye.

Later in 1925 the child began to have frequent convulsive attacks, without auras, but at no time did he froth at the mouth, bite his tongue or injure himself elsewhere. During the following summer there was a constant running of clear fluid from his nose, which was found to be cerebrospinal in origin.

As the convulsive attacks became more frequent, he was admitted to the Training School at Vineland, N. J., and later was transferred to the Village for Epileptics at Skillman, N. J. (September 1936), when 25 years of age. In November 1937, during an altercation with another patient in his cottage, he either fell or was knocked down by the other patient, and his head struck the floor. He was unconscious for about ten minutes and then complained of dizziness and headache and soon became nauseated and vomited. There was no evidence of fracture of the skull, but a week later he became more confused, and the

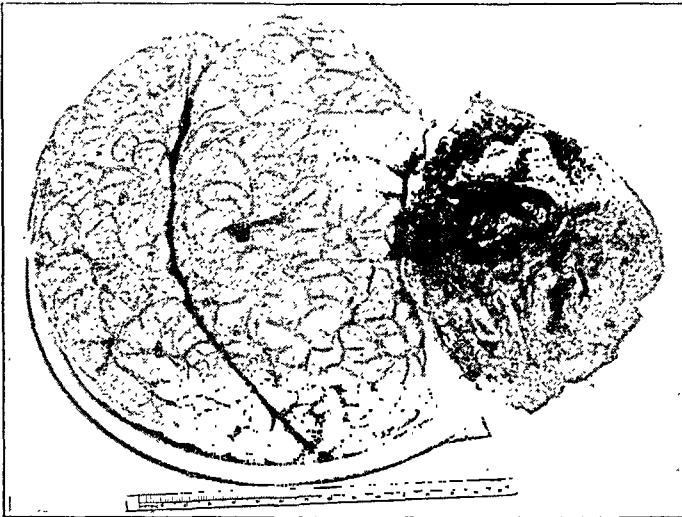


Fig. 1.—Appearance of the brain with a hematoma.

temperature rose to 104 F. as the result of a pleural pneumonitis at the base of the right lung. He was sent to the infirmary, and on December 14 respiratory distress and cyanosis suddenly developed. Acute massive pulmonary edema developed, and death occurred within two hours.

The body was sent to the University Hospital, and a complete autopsy was performed by Dr. B. J. Alpers. His report follows:

"The brain was huge, weighing 2,200 Gm. Part of the large size was the result of internal hydrocephalus, since the cerebral hemispheres were fluctuant. Most of it, however, was due to an actual increase in size of the brain, a true megalencephaly. The cerebral hemispheres were about one third larger than normal; the cerebellum and brain stem were proportionately enlarged.

Over the left cerebrum was a large subdural hematoma which covered the frontal and part of the parietal lobe. It measured 1 cm. in its thickest portion. The clot was dense and firm and appeared old; it was adherent to the overlying dura.

"The gyri were larger than normal, but there was no difference in the consistency of the gyri. They felt quite normal. Small areas of suffusions were seen over the inferior surface of the right temporal lobe. The brain in general

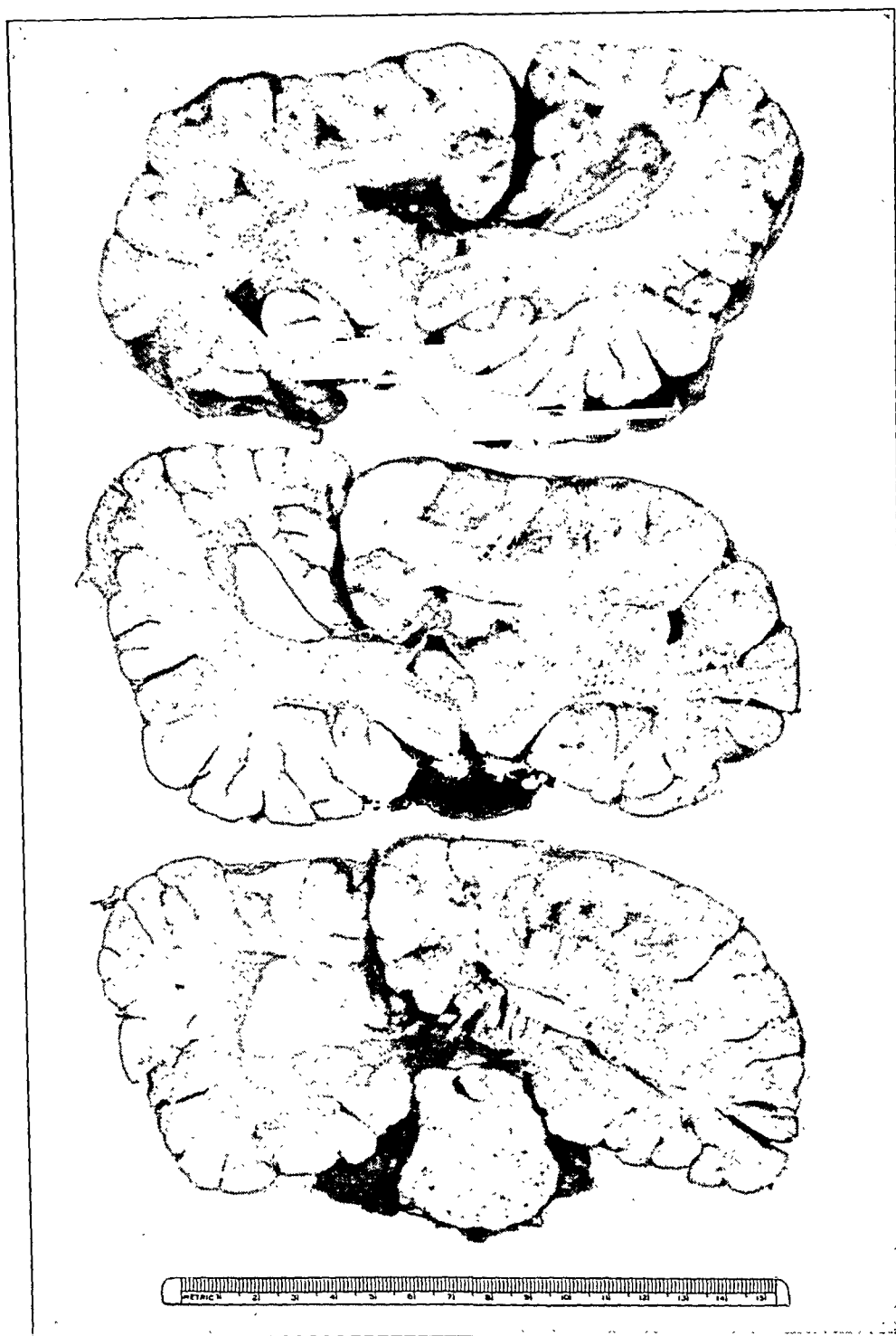


Fig. 2.—Tumor of the third ventricle, extending through the aqueduct of Sylvius into the fourth ventricle.

had a cyanotic appearance. The meninges over the base were thickened and contained blood.

"Section of the brain revealed large gyri with a large amount of white matter. Here and there, in the white matter chiefly, but also in the cortex, were small pink hemorrhagic areas. Most of these were in the left cerebral hemisphere, especially in the left frontal region. A few such areas were seen in the left putamen and several in the right parietal region. These looked like fresh areas of metastatic encephalitis.

"Filling the third ventricle was a small tumor which extended from the posterior part of the third ventricle through about the middle of the pontile portion of the fourth ventricle. This tumor was adherent to the floor of the third ventricle and to the right lateral wall of the pons. It had no relation to the pineal body. It was firmly adherent to the ependyma and seemed to arise from this structure. It did not fill the third ventricle completely, there being about 3 mm. between the top of the tumor and the tela choroidea. The tumor was white, soft and relatively avascular. Beneath it, on the left side of the mesencephalon, was a linear softening extending under the floor of the aqueduct and along the raphe to the interpeduncular space. The softening was old and contained much hemosiderin pigment.

"There was marked internal hydrocephalus involving the lateral ventricles. The ventricle behind the block (the fourth ventricle) was not dilated. The anterior horns and atrium were much more affected than the inferior horns. The corpus callosum was thin, and the basal ganglions were much compressed by the dilated ventricles."

Dr. Alpers summarized the case as one of a large brain with a tumor of the third ventricle, internal hydrocephalus, subdural hematoma and many foci of metastatic encephalitis.

The gross diagnosis was: "(1) tumor of the third ventricle; (2) subdural hematoma on the left side; (3) megalencephaly; (4) internal hydrocephalus, and (5) metastatic encephalitis."

SUMMARY

A case of chronic internal hydrocephalus in a boy 10 years of age is reported. Associated symptoms were papilledema, with failing vision and contracted visual fields, and partial oculomotor involvement. Decompression brought about relief of the intracranial pressure, with improvement of vision and widening of the fields. Gradual mental deterioration, spells of unconsciousness and later convulsive attacks occurred. There was rhinorrhea due to erosion and perforation of the cribriform plate. Death occurred at 27 years of age from extensive subdural cerebral hemorrhage due to accidental traumatism. Autopsy showed the "presence of an infiltrating tumor (astrocytoma) of the hypothalamus and the third ventricle, which extended through the aqueduct of Sylvius into the fourth ventricle, partially blocking the out-flow of ventricular fluid, and metastatic hemorrhagic encephalitis of the cerebral cortex."

The surgical aspect of the case will be discussed by Dr. Francis C. Grant, who followed the case with Dr. Frazier for years, and the neurologic point of view by Dr. B. J. Alpers, who made the postmortem examination.

DISCUSSION

DR. FRANCIS C. GRANT: From the surgical standpoint, one speculation at least is of interest. The boy came under neurosurgical observation in 1921. The clinical signs were few and no positive localization of the position of the lesion producing a mild bilateral choking of the optic disks could be made. Ventriculography was not highly regarded at the time. No ventriculograms, which in all probability would have shown the position of the tumor, were made. But even if a localization could have been reached by this method, the operative approach to a tumor in the third ventricle was then, and for that matter still is, a hazardous procedure.

Could any operative attack then or now directed against an astrocytoma adherent to the floor and the ependyma of the third ventricle have assured the patient of a survival period of seventeen years? The simple, easily performed, suboccipital craniectomy, done then in ignorance for relief of pressure, accomplished an amazingly satisfactory result. Of course, the slow growth of the tumor which permitted the cerebrospinal fluid to force its way around this obstruction in the third ventricle was a fortunate circumstance. Nowadays no neurosurgeon would consider a suboccipital craniectomy for relief of pressure when the tumor was known to be in the third ventricle. Through the occipitoparietal approach, either by working through the ventricle following a transcortical incision or by mobilization or amputation of the right occipital lobe, the corpus callosum would be reached and transected and the tumor removed. With such a procedure the child would have had a 30 to 40 per cent chance of immediate death from hyperthermia or hemorrhage. If he had recovered, a marked defect of the visual field would have resulted, with possibly hemiparesis from unavoidable section of the rolandic vein. At times the valor of the modern neurosurgeon outruns his discretion. This case is important because it emphasizes the fact that a properly placed, simply performed decompression with consequent relief of pressure may produce as satisfactory results as a hazardous direct attack on the tumor.

Furthermore, in 1925, four years after operation, rhinorrhea developed. Dr. Frazier was away at the time, and the patient came under my care. No way of checking the leak seemed possible, and an attack of meningitis with fatality seemed inevitable. But within three months the rhinorrhea ceased spontaneously. Neither before nor after the leakage of spinal fluid was there any change in the clinical signs or ophthalmoscopic findings.

Convulsions then set in, and the patient was hospitalized, death finally occurring from a chronic subdural hemorrhage. If one should care to philosophize on this case, it is curiously tragic that the patient should have carried a probably inoperable tumor for seventeen years and survived a rhinorrhea which surgical intervention could not stop, only to die of a subdural hemorrhage, a lesion which is easily and safely removed by a simple surgical procedure.

DR. B. J. ALPERS: From the standpoint of pathology, I think that it is well to point out the fact that a great many of these tumors that are spoken of as tumors of the third ventricle are not really such. They are tumors which involve the adjacent brain substance and belly out

into the third ventricle. Only from that standpoint should they be spoken of as tumors of the third ventricle.

A good deal of the confusion that has resulted with regard to tumors of the third ventricle has come from regarding these infiltrating tumors which project into the third ventricle as really being tumors of the third ventricle. Actually, primary tumors of the third ventricle are rare. There are some colloid tumors which fill the third ventricle; they are really unusual. Rarely does one see dermoid cysts or other cysts arise primarily in the third ventricle. I think that if one considers the primary tumors of the third ventricle, the matter becomes simplified from the pathologic standpoint but confused from the clinical standpoint. In these cases there is a history of increased pressure and possibly of intermittent headache and, as Oldberg and Eisenhart have pointed out, a history in a small percentage of cases of personality and emotional disturbances. I do not think it is possible to make more than a good guess at a diagnosis of a tumor of the third ventricle in most instances. In cases in which there is increased intracranial pressure without localizing signs, one is entitled to a guess of tumor blocking the ventricle.

PRIMARY TUMORS OF THE OPTIC NERVE (A PHENOMENON OF RECKLINGHAUSEN'S DISEASE)

A CLINICAL AND PATHOLOGIC STUDY WITH A REPORT OF
FIVE CASES AND A REVIEW OF THE LITERATURE

FREDERICK ALLISON DAVIS, M.D.

MADISON, WIS.

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INTRODUCTION AND CLASSIFICATION

Primary tumors of the optic nerve are rare. Collins and Marshall reported only 2 over a period of fifteen years among 388,000 patients treated at the Royal Ophthalmic Hospital (Moorfields) in London. Verhoeff reported that 4 primary tumors of the optic nerve were removed at the Massachusetts Charitable Eye and Ear Infirmary over a period of thirty-six years, during which time 669,557 new patients were examined. In these two large series of patients with ocular conditions the tumors occurred in the ratio of about 1 in 176,000 patients. Three hundred cases were collected from the world literature by von Hippel and were cited in the 238 references in the bibliography of this author's exhaustive monograph in the 1925 edition of Graefe and Saemisch's "Handbuch der Augenheilkunde." I have found 65 references to this subject since that date, with a report of 80 additional cases. Much confusion is still evident in this material, but considerable progress has been made in the histologic diagnosis and classification.

Since the publication in 1912 of Hudson's classic paper concerning primary tumors of the optic nerve, most authors, with the exception of those of the French school, some Spanish observers and a few others, agree that these tumors may be classified into three main types according to their histologic nature as follows: (1) those characterized by gliomatosis (glioma), which arise within the nerve stem; (2) endotheliomas, which arise in the arachnoid or the dural sheath, and (3) those characterized by fibromatosis (fibroma), which arise in the dural sheath. The tumors have been further classified according to their location in the nerve. The terms "subdural" and "extradural" have been most generally employed, since they denote the position of the growth with relation to the outer, or dural, sheath. The first two types, namely, the glioma and the endothelioma, are subdural, while the fibroma is classed as extradural, since it usually appears to grow from the outside of the dural sheath.

Verhoeff accepted this general classification but suggested the terms "intraneural" (for tumors arising in the nerve stem) and "extraneural" (for tumors arising in the sheath). He further advocated the term "glioma" in place of "gliomatosis." The former term has also been used by others.

In the cases reviewed by Hudson, as well as in those more recently tabulated by Lundberg, by far the greater number of the tumors were glial in nature, though a number of endotheliomas were also reported. Fibromatosis of the nerve sheath is exceedingly rare, few cases having been recorded.

From the Department of Ophthalmology, University of Wisconsin Medical School.

Read before the Section on Ophthalmology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 18, 1939.

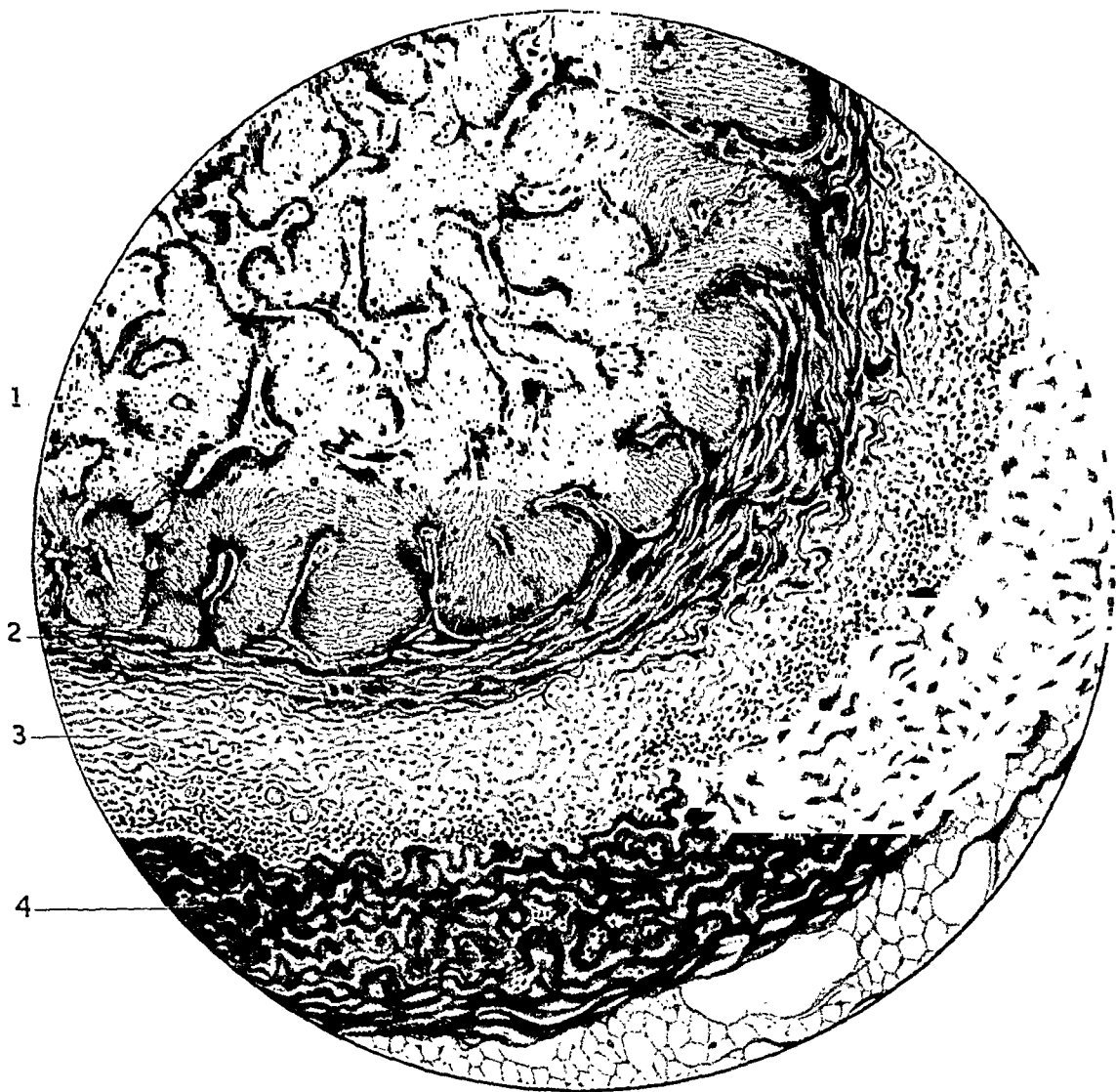


Fig. 1.—Glioma of the optic nerve in a comparatively early stage (stage 2) of development showing: 1, proliferation of the glia cells of the nerve stem, chiefly fibrous astrocytes; 2, a neoplasm invading the pial sheath; 3, hyperplasia of mesothelial cells of the subdural and subarachnoid spaces; 4, dural sheath (thickened); Mallory's phosphotungstic acid-hematoxylin stain.

The clinical signs of tumor of the optic nerve are well known and require little comment here. The outstanding features are painless unilateral exophthalmos, which progresses slowly, accompanied by choked disk or simple atrophy of the optic nerve, with marked reduction in visual acuity. A roentgenogram may show enlargement of the optic foramen. In the early stages the eye usually projects directly forward, with but slight limitation of motion, though this is variable. Upward rotation is usually the first to be restricted or lost. At times there is a tendency for the globe to be directed slightly downward, inward or outward, depending on the duration and the size of the growth. Ptosis may be present. Examination of the fundi may also show white spots surrounding the disk and scattered about the retina, especially toward the macula. If the choking is great, there are occasionally hemorrhages.

The diminution in visual acuity generally develops earlier in cases of glioma than in cases of endothelioma due to the involvement of the nerve stem proper in the former, while in the latter the nerve suffers later through compression.

The glioma is usually seen in the first decade of life and is probably congenital in most cases. Hudson reported that nearly 75 per cent occurred before the tenth year, though some are found in later life. The endothelioma is more frequently seen in older subjects, 50 per cent of the patients in Hudson's series being over 30 years of age. Early removal of the growth by operation is universally advised.

REVIEW OF THE LITERATURE

It is not intended in this paper to present an exhaustive review of the literature concerning tumors of the optic nerve in general, since this has been so thoroughly and ably presented by Byers, Hudson and von Hippel and more recently by Lundberg. Though some of this material has been reviewed, it would be useless repetition to enumerate all the cases cited by them.

Hudson reviewed 182 case reports up to 1912, and Lundberg tabulated an additional 123 from 1912 to 1934, making a total of 305 cases.

A summary of the pathologic diagnoses in these combined surveys reveals the following figures:

Gliomatosis (glioma)	193 (probable and certain)
Endotheliomas	86 (probable and certain)
Fibromatosis of the sheath.....	8
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Twenty-nine cases have been recorded since the publication of Lundberg's monograph. Also, there are a number of case reports in the literature not included in either of the foregoing surveys. It appears, therefore, that approximately 380 cases are on record. Reference to a

few of the more complete surveys of the subject reveals how opinions regarding the histologic structure of the growths have changed from time to time. Most of the early investigators regarded the tumors as mesoblastic in origin and designated them as fibromas, fibrosarcomas and myxomas, though there are some early cases in which a diagnosis of glioma was made.

Byers' monograph in 1902 apparently exerted considerable influence, for his conclusions concerning the histologic structure of the tumors received wide acceptance. Reference to some of the standard textbooks reveals that a number of authors still endorse his point of view. He made a complete study of the literature on the subject and tabulated 102 case reports, to which he added 2 of his own. In 1 of these cases (case 2), however, no microscopic studies were made of the original tumor, since it had been removed nine years previously, and the specimen, according to the author, was dry and unfit for study. The patient survived nine years, and on death autopsy by Adami revealed a "psammoma-sarcoma" of the brain. In the other case (case 1), reported also by Buller in 1886, a tumor of the optic nerve had been removed from a child of 7, but a complete anatomic description of the growth was not made until fifteen years later, the specimen presumably having been in Müller's fluid during that period. For the microscopic study of sections of this tumor, the author mentioned the use of the methods of staining of Unna, Weigert, Pal-Weigert and Van Gieson. Briefly summarized, the description of the growth follows: ". . . The enlargement in the nerve was due to an increase in the intrafascicular connective tissue (endoneurium) which formed a fine honeycombed fabric, the cells of which as shown by Van Gieson and Pal-Weigert were found to be entirely free from any nerve elements proper." He also stated that the tumor of the nerve sheath was composed of "irregular flowing masses of connective tissue, which formed a meshwork of varying coarseness." He considered the growth to be primary, the tumefaction being a simple hyperplasia of all the connective tissue structures of the nerve; the sensory elements were entirely absent, and dilatation of the lymph spaces of the nerve stalk was noted.

Byers concluded from this study and his critical review of the literature that "indefinite as the term fibromatosis may be, it well defines in a general way all the changes that occur in the tumors under discussion." The tumors in 10 of 100 cases, however, in which he tabulated the anatomic diagnosis given by authors recording them, were reported as some form of glioma, gliomyxoma and gliomyxosarcoma. He considered the tumor of the nerve but a part of a neoplasm more or less widely affecting other structures within the skull.

Parsons (1905) made an extended reference to Byers' review in his textbook, "The Pathology of the Eye." In this work he stated that there is little evidence in favor of specific neuroglial proliferation in these tumors, though this occurs as a subsidiary phenomenon. He agreed with Byers that the growths are essentially of mesoblastic origin.

Hudson (1912) rejected the findings of a majority of investigators to that date and concluded that most of the primary tumors of the optic nerve arise from the neuroglia within the nerve stem. He based his conclusions on a study of 183 cases collected from the literature and 3 cases which he reported. He stressed the fact that the overgrowth of glial tissue is the essential feature of these tumors, though many authors had not recognized this. Hudson considered the growth a "degenerative gliomatosis," following the term "gliomatous degeneration" suggested in a case report by Fischer. He placed considerable emphasis on the fact that no recurrence of the new growth in the orbit had been recorded. The term "degenerative gliomatosis" implies "a generalized overgrowth of neuroglial tissue of infiltrative character dependent on some degenerative change in the tissues of unknown etiology." He remarked, however, "that in many, if not the majority, of cases it [the new growth] is not limited to the orbital portion of the nerve, but extends into the intracranial cavity more or less extensively." Hudson attributed the recognition of the real nature of the tumors to Leber and Willemer and stated that the tumor tissue is characterized by cells first described by Vossius and Salzman. He listed the tumors in 29 of the cases he reviewed, however, as true endotheliomas, with those in an additional 28 as probably endotheliomas.

Verhoeff (1922), in an extensive analysis of this subject before the American Ophthalmological Society, based on a histologic study of 11 tumors, confirmed the findings of Hudson and, as noted previously, advocated the classification "intraneural" and "extraneural." A further communication by Verhoeff (1932), in which he reported a study of 12 additional tumors, substantiated his view that "almost all, if not all of the primary intraneural tumors of the optic nerve described in the literature are gliomas."

Von Hippel (1925), in the monograph cited in the opening paragraph of this paper, concluded that most tumors of the optic nerve originate from glial tissues, though some originate from the arachnoid membrane. He suggested that there is probably an inherent growth tendency in both glial and mesodermal tissues. He considered proliferation of the glia the most important factor in this type of tumor but noted that in many cases there is also proliferation of connective tissue within the nerve sheath, which proliferation is of so great a degree that, although intermingled with the glia, it cannot be overlooked in characterizing the tumor. He doubted that it is entirely justifiable to designate such tumors as "glioma" and suggested the term "fibrogliomatosis," which indicates that proliferation of both ectodermal and connective tissue occurs.

Oberling and Nordmann (1927) offered a different and original classification of tumors of the optic nerve based on the studies of Oberling. They stated that in general the characteristic cells of most of the tumors belong to the leptomeninx, which are special cells that are distinct from

endothelial cells, which they called meningioblasts, though they described tumors arising from the glial elements of the nerve as well. These meningioblasts are presumably the normal mesothelial cells which line the subdural space. They frequently appear on the outer surface of the arachnoid membrane, grouped in clusters or nests. The neoplastic cells, according to these authors, show finely fibrillar acid-staining cytoplasm, with an oval or round nucleus containing fine granular chromatin, enclosing at times a large opaque nucleolus. The nucleolus can become transformed into a vacuole, which then occupies a large part of the nucleus. These cells vary in their arrangement, according to the authors:

At times they are in nests like epithelial cells; again they are vaguely polyhedral and fusiform. At times they arrange themselves in whorls, in the center of which appear collagenous spheres. The structure of their cytoplasm gives them a neurogenic appearance, while their polyhedral form resembles epithelial cells; the whorls, made up of fusiform cells, resemble fibroblasts of fusiform sarcoma.

Oberling and Nordmann expressed the opinion that these meningioblasts are cells of neural origin which have wandered into the perineural mesenchyme and that they are homologous to the cells of Schwann. They therefore identified three systems of neuroglia: (1) central glia, (2) glia of the nerves formed by the cells of Schwann and (3) meningioblasts. They suggested a comprehensive classification of tumors of the optic nerve based on the neuroepithelial theory of Oberling.

Satanowsky and Androgué (1929) accepted the interpretation of Oberling and Nordmann and rejected that of von Hippel. They insisted that most of these tumors arise from the meninges, especially the arachnoid, and considered them to be meningioblastomas. They reported 6 cases, with detailed histologic descriptions.

Many other authors, particularly from the French school, followed the lead of Oberling and Nordmann and therefore adopted the term "meningioblastoma" in describing these tumors.

Lundberg (1934), in the monograph previously referred to, presented a historical review of the subject and reported 10 cases of primary tumor of the optic nerve and chiasm from the Caroline Institute of Stockholm.¹ Nine tumors were reported as gliomas of the oligodendrocytoma type and 1 as a meningiopsammoma. This is one of the most exhaustive studies of the subject that has yet appeared and should receive serious consideration of workers in this field.

The statistical data now available reveal that the tumors in nearly two thirds of the cases on record are predominantly glial in nature, while about one-third may be grouped under the classification "endothelioma."

1. I studied some of these sections during a recent visit to the Caroline Institute, by permission of Dr. Lundberg and Professor Henschen.

That disagreement in terminology still prevails is further shown by brief reference to some of the standard textbooks.

Parsons' text (1927) retains the classification "intradural" and "extradural" tumors. He stated that "they (the former) rarely spring from true nervous tissue (gliomata—resembling cerebral gliomas) but usually originate in the connective tissue septa derived from the pia mater and from the arachnoid sheath." He regarded most of these and the extradural tumors as probably endotheliomas.

In Friedenwald's "The Pathology of the Eye" (1929) tumors of the optic nerve are divided into two groups: (1) those arising in the nerve sheath, which are designated "neurofibroma" and "neuroma," and (2) gliosarcomas of the meninges, which, according to the author, "grow down the sheath of the nerve by simple extension from the cranial cavity."

In the tenth English edition of Fuchs's "Diseases of the Eye" (E. V. L. Brown translation) it is stated:

Primary tumors of the orbital portion of the optic nerve are predominantly of two kinds:

1. Endotheliomata, which develop from the dural sheath.

2. Those which develop from the subarachnoidal trabeculae; from the pial sheath; from the septa; from the supporting tissue of the optic nerve and, according to their histologic make-up, are called: fibroma, sarcoma, myxoma, myxosarcoma, and glioma.

However, despite all this, they all belong to one form and only represent varying forms of neurofibromatosis.

Collins and Mayo in their "Pathology and Bacteriology of the Eye" (1925), van der Hoeve in Beren's "The Eye and Its Diseases" (1936), Verhoeff in Penfield's "Cytology and Cellular Pathology of the Nervous System" (1933) and Rhea in "Neuro-Ophthalmology" (1939) follow in the main the classification of Hudson.

It is evident from the literature cited that there is still some difference of opinion concerning the nature of primary tumors of the optic nerve. When one carefully studies the numerous detailed histologic descriptions which are on record, particularly those of tumor tissue for which the modern differential staining methods have been used, the tumors appear to fall in two main groups, namely, gliomas and endotheliomas (meningiomas), the former being much the more commonly encountered. The terms which have been commonly employed to designate the site of the growths, namely, "intradural" (subdural) and "extradural," as well as "intraneural" and "extraneural," appear equally unsatisfactory to me.

Both gliomas and endotheliomas are subdural (intradural) and both are also usually extraneural. A glioma starts as an intraneural growth but almost invariably later penetrates the pia and grows in the inter-

vaginal spaces, where it is no longer strictly intraneural but has become extraneural or subdural. Likewise an endothelioma may be extradural as well as intradural (subdural), depending on the seat of origin. I therefore believe that the terms should be abandoned since they are too confusing and often inaccurate.

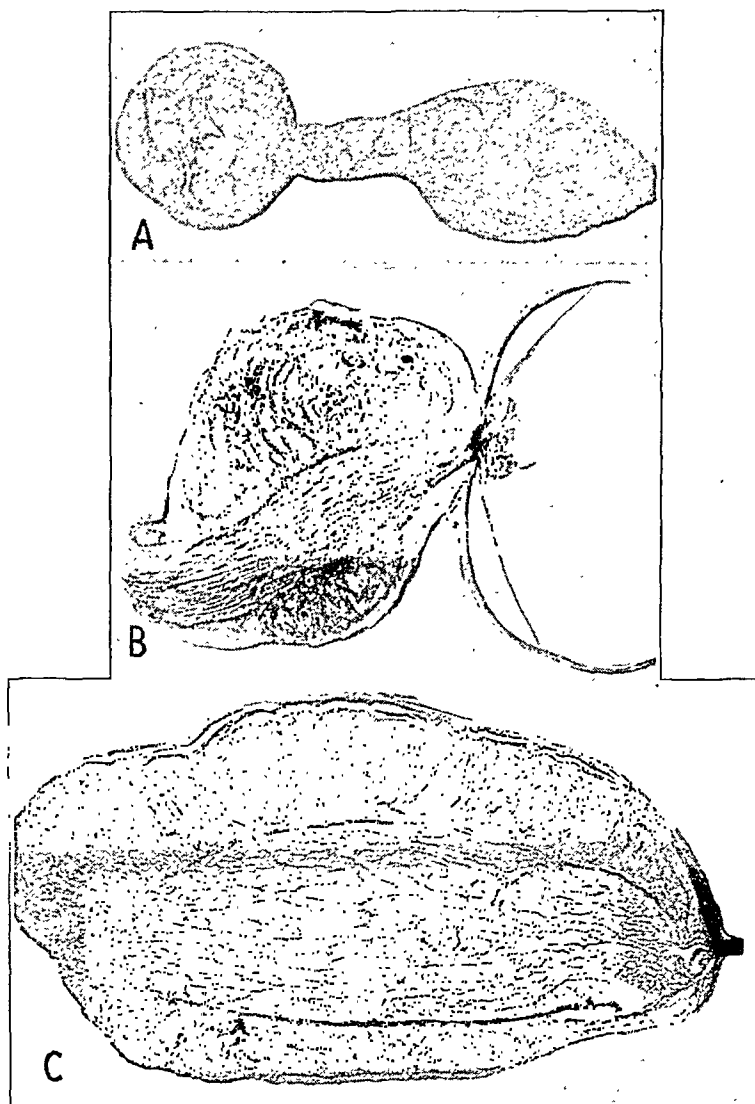


Fig. 2.—*A*, glioma of the optic nerve. *B*, glioma of the optic nerve showing cystic and gliomatous involvement of the optic disk. (*A* and *B* are from Verhoeff, F. H.: *Primary Tumors of the Optic Nerve*, in Penfield, W.: *Cytology and Cellular Pathology of the Nervous System*, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1029; *Primary Intraneural Tumors of the Optic Nerve*, *Arch. Ophth.* **51**:120, 1922.) *C*, glioma of the optic nerve. (From Reese, R. G.: *Primary Intradural Tumor of the Optic Nerve*, *Arch. Ophth.* **49**:515, 1920.)

Gliomas.—Gliomas (fig. 2) arise within the nerve stem from abnormal proliferation of the neuroglial cells. Since the nature of such tumors

and their relation to Recklinghausen's disease are the theme of this paper, a more complete description of them will be deferred until after the case reports.

Endotheliomas.—Endotheliomas (meningioma and arachnoid fibroblastoma) involve the sheath of the nerve, the nerve stem proper being affected only indirectly from the pressure (fig. 3). Histologically this type of tumor is characteristic. The general structure is entirely different from that of the glioma. As a rule the tumor is made up of masses of cells (fig. 4) resembling those of the "arachnoid clusters," or cell nests, which are normally seen on the outer aspect of the arachnoid membrane. They appear in closely packed whorls or bundles with relatively little intercellular connective tissue, though this appears at times in the septums which divide these masses of cells. The nuclei are large and oval, staining faintly with hematoxylin. Whorls of hyalinized



Fig. 3.—Psammoendothelioma of the optic nerve. (From de Schweinitz, G. E.: A Contribution to the Subject of Tumors of the Eyelid and Orbit [case 3], Tr. Am. Ophth. Soc. 14:341-355, 1915.)

connective tissue, hyalinized blood vessels and corpora arenacea or psammoma bodies are frequently present. The tumor appears to spring from the arachnoid sheath as a rule, though it often invades the dural sheath of the nerve. At times it arises from the dural sheath itself (Benedict, Parker, Stephenson) (fig. 5). While it does not invade the nerve stem, it frequently spreads outward, through the dura into the orbit. It resembles an intracranial tumor of the type described as meningioma (psammoma type) by neurosurgeons and neuropathologists.

The origin of such tumors is still a controversial subject. Whether they are truly primary growths which arise within the intraorbital part of the sheath of the nerve or merely extend into it from a similar growth within the cranium is not always clear. Verhoeff emphatically stated that an endothelioma arising from the intraorbital portion of the optic nerve has not yet been demonstrated. Other writers evidently do not accept this point of view.

Unquestionably many of these tumors arise by direct extension from intracranial meningiomas, as in the case reported by Worster-Drought, Dickson and McMenemey. However, since intracranial meningeal tumors are at times multiple, there seems to be no valid reason why they should not also appear as primary growths in the arachnoid or the dural sheath of the optic nerve. The growth in a case reported by Parker and Benedict, with an autopsy report by Kernohan and Parker some years later, appears to have been of this nature. In this case

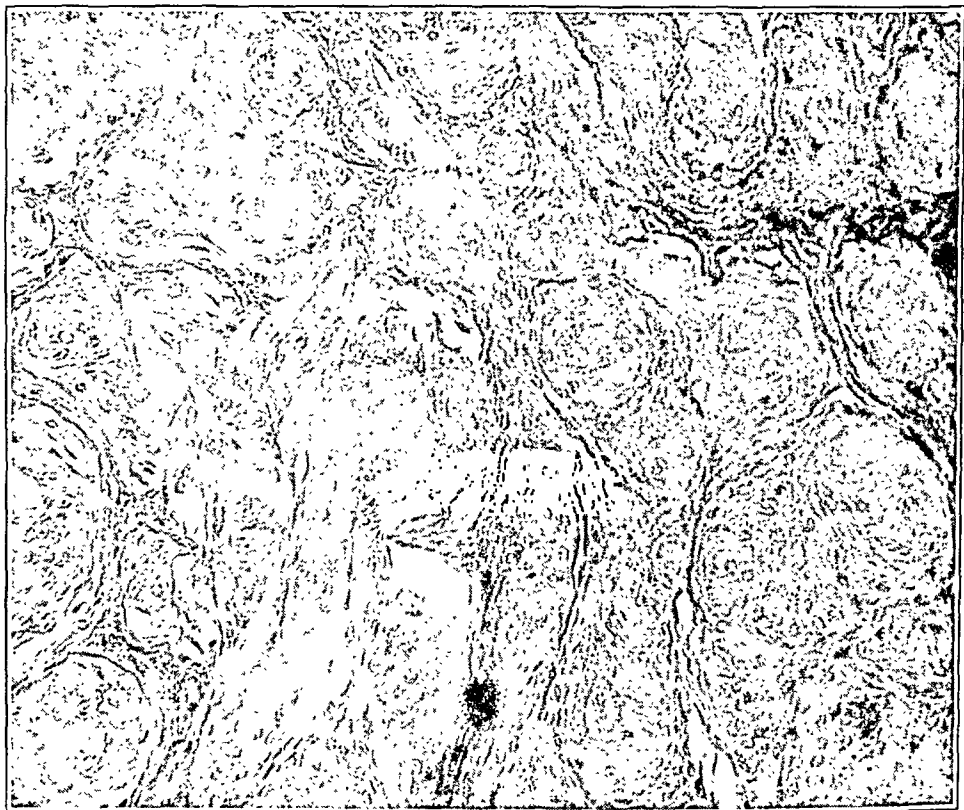


Fig. 4.—Cell nests in an endothelioma of the arachnoid sheath (arachnoid fibroma). (Photograph of a section supplied by Dr. E. L. Goar; unpublished case.)

no local recurrence appeared in the orbit after removal of the tumor, nor was there a growth found within the vicinity of the optic foramen at autopsy, although an independent growth was present elsewhere within the skull. (A more detailed report of this case is given in a later section.^{1a})

It seems possible that these tumors may arise from cell nests embedded in the dural sheath of the nerve, similar to those which appear in the cranial dura, as suggested and described by Weed. I

1a. This will be found in the concluding part of this article, in the May issue, in the section on "Relations of Optic Nerve Tumors to Recklinghausen's Disease."

have on several occasions observed similar nests of cells embedded in the dural sheath of normal optic nerves.

An endothelioma of the optic nerve usually appears after the second decade of life, according to Hudson. It grows slowly and appears relatively benign. In some of the cases the patients have been followed for ten to fifteen years after operation without evidence of local recurrence in the orbit. As pointed out earlier, however, further development of similar lesions within the cranium may follow, resulting in death.

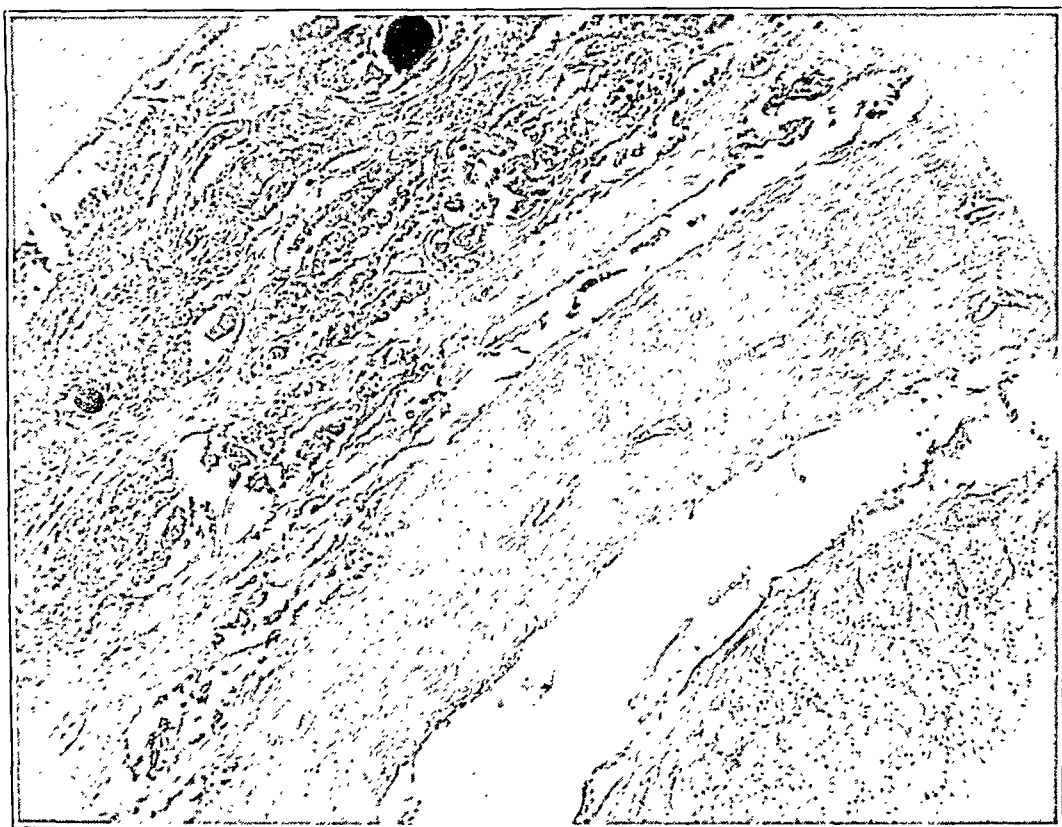


Fig 5.—Endothelioma of the optic nerve. The tumor (upper left side of illustration) arose from the dural sheath of the nerve. Note the two psammoma bodies. The nerve (lower right side) was not involved. (Photograph of a slide sent by Dr. W. L. Benedict.) Descriptions of this tumor appear in the reports of Benedict (*Am. J. Ophth.* **6**:182-201, 1923) and of Parker (*J. Nerv. & Ment. Dis.* **56**:441-452, 1922).

[Since this paper was completed, my attention has been directed to Cushing and Eisenhardt's recently published book, "Meningiomas." It contains a most illuminating chapter concerning meningiomas of the sheath of Schwalbe and a wealth of information concerning related subjects. The question of whether or not an endothelioma of the optic nerve sheath is primary in origin or merely an extension down the vaginal sheath from a similar tumor within the cranium is discussed at some length. In a postscriptum they cited several cases from the

literature—Shapland and Greenfield's (cited in a later section^{1a}), Caston's and Friedenwald's (unpublished)—in which the tumors were apparently primary in origin. The authors stated that in the case of Friedenwald the proximal stump of the nerve adjacent to the foramen showed normal uninvolved nerve and nerve sheath. A roentgenogram showed a normal foramen. They concluded with the statement: "This case appears definitely to settle the debatable point we have raised."

The one example of a meningioma of the optic nerve sheath in Cushing and Eisenhardt's own series, however, arose by extension from a meningioma of the olfactory groove (fig. 6). These authors directed attention to the possibility of orbital extension by direct invasion from meningiomas which arise from various parts of the sphenoid ridge, though suprasellar tumors arising in the region of the optic foramen never seem to invade the vaginal sheath. They suggested, therefore, that the locus of origin for these tumors may be in the foramen itself and that the tumors may as well grow backward into the cranial cavity as forward down the sheath.]

Fibromatosis.—Fibromatosis of the optic nerve sheath is so rare that it has been little studied. Hudson cited but 7 cases in his review. Dr. E. L. Goar has made it possible for me to study sections from a tumor (fig. 7), the report of which is as yet unpublished. The dense connective tissue which fills the orbit is intimately fused with the dural sheath and cannot be distinguished from it. It does not involve the arachnoid, nor is the nerve invaded. Verhoeff suggested that these growths may be residual changes which follow certain inflammatory masses within the orbit. Parsons classified these tumors as fibrosarcomas of a low grade of malignancy.

REPORT OF CASES

The patients in the 5 cases herein reported have been observed over periods ranging from three months to eight years. The patients in cases 1 and 2 were brother and sister, aged 4 and 6 years, respectively. The patient in case 3 was a child of 3 years. The tumor in this case was studied at two different stages of its growth, four and one-half years elapsing between examinations. The patients in cases 4 and 5 were women aged 20 and 27, respectively. The tumors in these 2 cases involved the chiasm and the adjacent nerves within the cranium, extending into the orbital part of the nerves. All 5 patients had a mild abortive type of Recklinghausen's disease, consisting of cutaneous café au lait-pigmented patches scattered over the trunk and extremities. These associated lesions were hereditary in 4 cases, though one generation was skipped in 1 case. A child of the fifth patient had similar peripheral cutaneous changes. One patient (case 3) also had deep-seated lesions within the brain as well as other peripheral changes in the nerves of the orbit and globe, while another (case 2) had osseous changes involving

the spine and fibula. In not 1 of the cases had a diagnosis of Recklinghausen's disease been made prior to this study, though most of the patients had been repeatedly examined. The presence of the tumors was verified by operation in all 5 cases, with autopsy in 1. Histologic

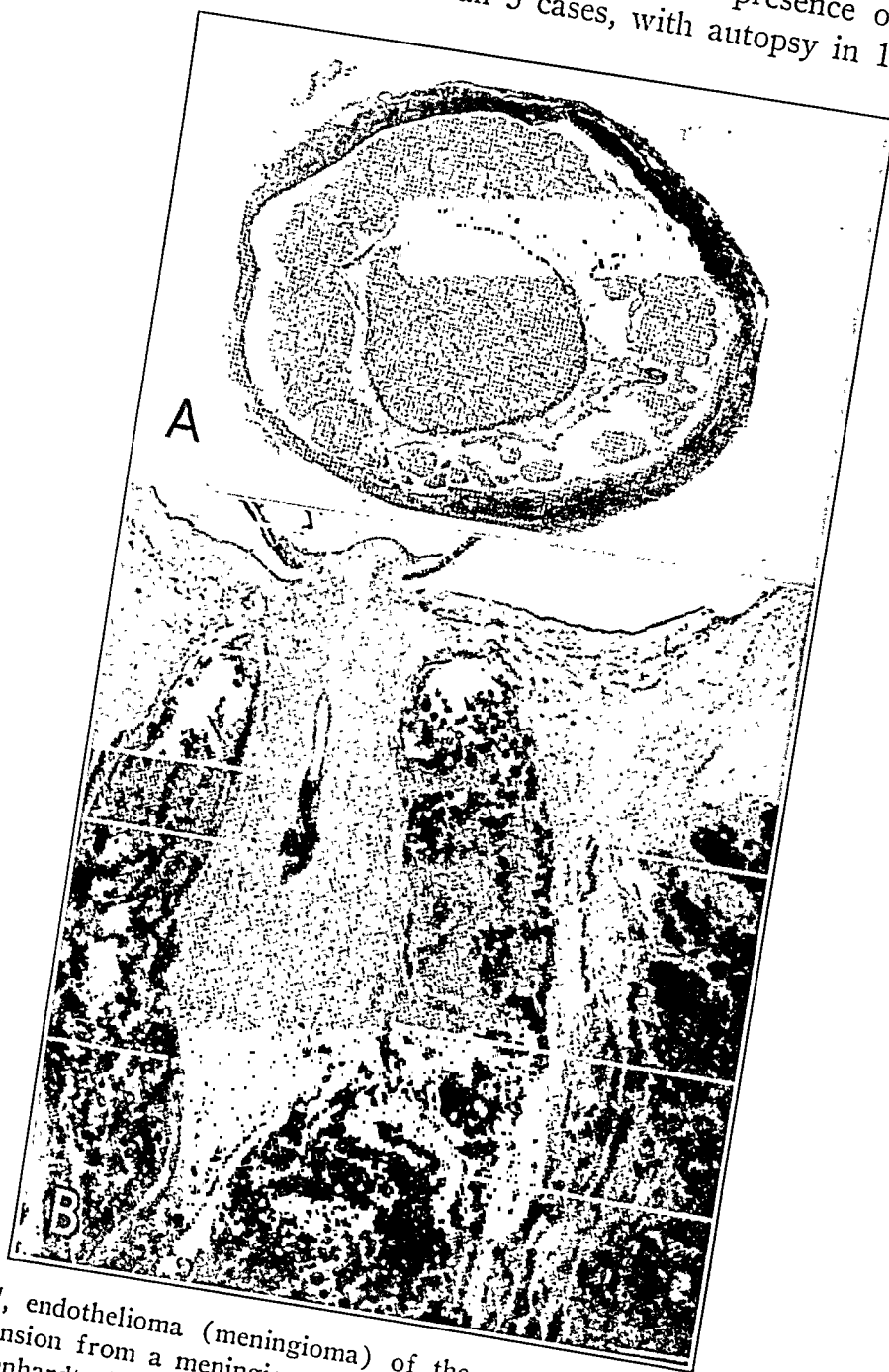


Fig. 6.—*A*, endothelioma (meningioma) of the arachnoid sheath. The tumor arose by extension from a meningioma of the olfactory groove. (From Cushing, H., and Eisenhardt, L.: *Meningiomas*, Springfield, Ill., Charles C. Thomas, Publisher, 1938.) *B*, longitudinal section of an endothelioma of the sheath of the optic nerve. (After de Leparsonne, from Cushing, H., and Eisenhardt, L.: *Meningiomas*, 1938.)

studies were made of the tumors in 3 cases. A diagnosis of glioma of the optic nerve was made in all 5 instances.

CASE 1 (fig. 8).—*Diagnosis of tumor of the optic nerve; Krönlein exploration followed by enucleation and exenteration of the orbit; glial proliferation of the nerve head, glioma of the optic nerve invading the sheath, peripheral Recklinghausen's phenomena—café au lait-pigmented patches; no recurrence; period of observation, eight years.*

H. H., a girl aged 4 years, was first admitted to the Wisconsin General Hospital on Oct. 16, 1931. The chief complaint was a protrusion of the left eye of six months' duration. Examination revealed exophthalmos, the exophthalmometer reading being 15 mm. greater for the left eye than for the right. The protrusion of the eye was for the most part directly forward, with slight downward and

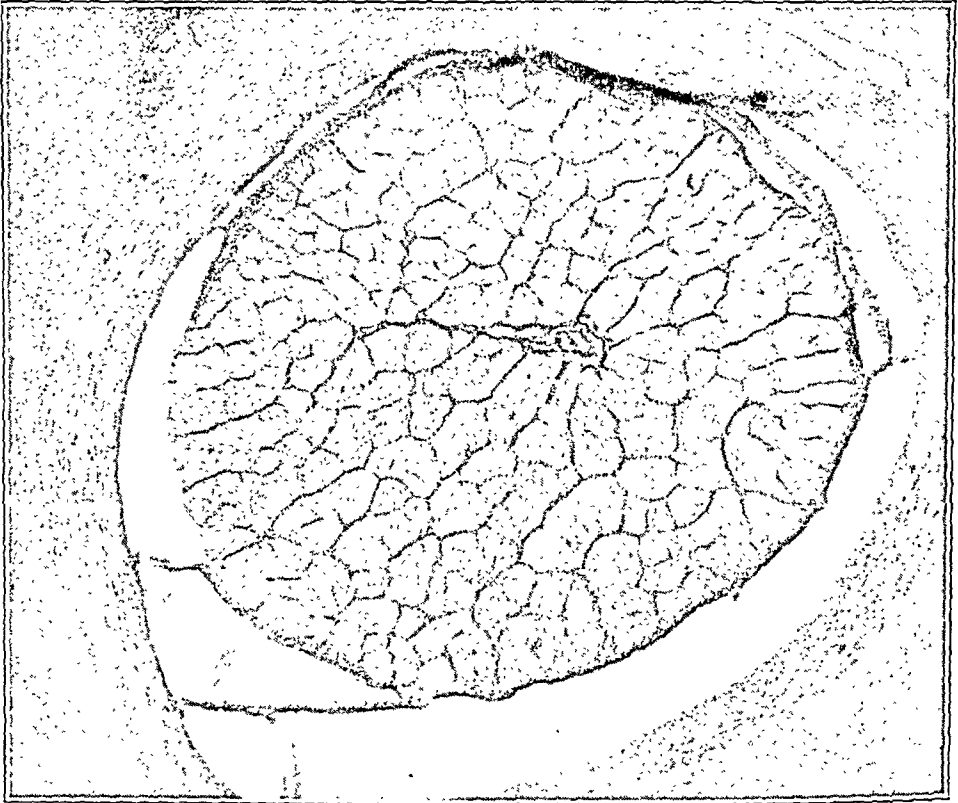


Fig. 7.—Fibromatosis of the dural sheath. Note the dense fibrous tissue which replaces the dura and extends outward into the surrounding tissues of the orbit. This is probably not a true tumor. Verhoeff suggested that it follows certain inflammatory masses in the orbit known as pseudotumors. Parsons referred to them as fibrosarcomas of low malignancy. Less than 15 such growths are on record in the literature. (Photograph of a section loaned by Dr. E. L. Goar; unpublished case.)

inward rotation. There was well marked ptosis. Motion of the globe was limited to downward and inward rotation. There were increased vascularity of the left upper lid and obliteration of the supraorbital fold, most marked in the outer portion of the lid. This fulness was soft and spongy, but no definite mass could be felt, nor was there a bruit. There was no bulbar or palpebral injection. The patient was unable to elevate the eye or to turn it outward. The pupil, somewhat

larger than that of the right eye, was eccentric and displaced nasally and downward. It reacted to light and dilated concentrically with atropine. Ophthalmoscopic examination showed the media to be clear. There was a definite swelling of the nerve head of about 4 diopters. The blood vessels were dilated and tortuous, with a few hemorrhages on the disk and about its margins. There was a whitish, ringlike band in the retina surrounding the disk, which was elevated. It appeared to be a low detachment or exudate under the retina. A white streak extended from this temporally and below the macula. Other scattered white spots suggesting exudate were seen in the region of the macula, and the entire temporal half of the retina appeared pale. The eye was apparently blind. A diagnosis of tumor of the optic nerve with possible extension into the globe was made.

The right eye was normal.

Previous Medical and Family History.—The child was one of twins. The previous medical history was unimportant, the child having had none of the diseases of childhood or any previous ocular trouble. The mother showed mild Recklinghausen's disease, consisting of numerous brownish café au lait patches on the skin

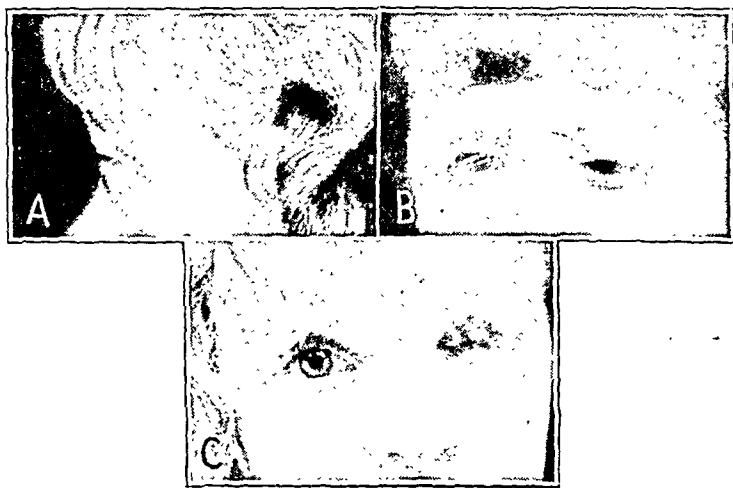


Fig. 8 (case 1).—*A* and *B*, front and side views of H. H. with glioma of the optic nerve. There is exophthalmos of the left eye of 15 mm. *C*, appearance of patient one year after operation (Krönlein exploration followed by enucleation and exenteration of the orbit).

of the trunk and extremities, with several soft tumor masses on the back, legs, abdomen and chest. Her face showed a number of small knoblike subcutaneous swellings, especially about the chin. Her eyes were normal. (Data were obtained after operation on the child.) The twin brother was normal, showing no ocular involvement and no other sign of Recklinghausen's disease. A younger brother, admitted to the ophthalmic ward seventeen months later, showed a gliomatous involvement of the optic nerve of one eye as well as peripheral signs of a mild type of Recklinghausen's disease.

General Examination.—The general examination was exhaustive and included a tuberculin test, a Wassermann test, a spinal puncture and neurologic study, but the results were negative aside from the presence of a number of light brown non-elevated spots scattered over the skin of the abdomen, back and legs (fig. 9). The skin over the trunk was thickened and dry. The child was under observation two months, because of an infection of the upper respiratory tract. Anteroposterior and

lateral roentgenograms and stereoroentgenograms of the skull showed no abnormal changes in the cranial bones or in the left orbit. Subsequent study of the films, however, revealed a definite change in the profile of the sella turcia. Lateral stereoscopic views showed an ovoid, pear-shaped shadow which extended from the upper anterior margin of the body of the sella turcica under the anterior clinoid process. This projection was similar to, though not as large as, that found in case 3.

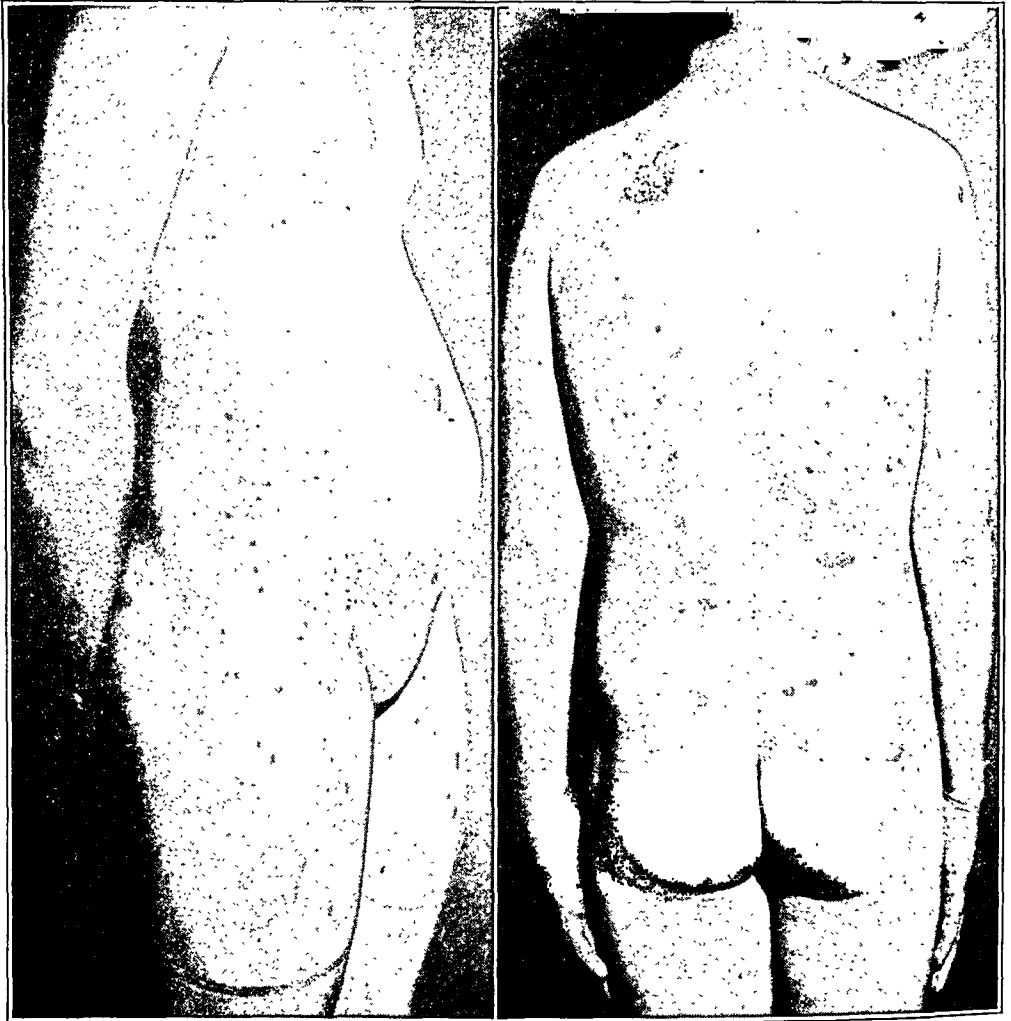


Fig. 9 (case 1).—Front and back view of patient taken several years after operation. Note the café au lait-pigmented spots. These were present at birth but increased in number and became more deeply pigmented.

Roentgenograms of the optic foramens were not made before operation. Later studies were not entirely satisfactory, but there appeared to be an elongation in one axis. The foramen of the right orbit was clearly outlined and appeared normal. The skull showed no evidence of intracranial pressure.

Operation (Dec. 16, 1931).—With the patient under anesthesia produced by avertin with amylene hydrate supplemented by nitrogen monoxide, the orbit was first explored through a Krönlein incision, and an encapsulated sausage-shaped

swelling of the nerve was found. A small incision was made in the dural sheath, and a cellular soft tissue exuded. Since a malignant growth was suspected, complete exenteration of the orbit was done and the Krönlein incision sutured. Recovery was uneventful, with the usual deformity.

Macroscopic Examination.—The globe, with contents of the orbit, was photographed, but unfortunately no view was taken after the removal of the soft parts.

The tumor involved the entire nerve in varying degrees and was completely encapsulated by the dural sheath, though the latter was much thinned in places. It was somewhat spindle shaped, being smaller at either end. The globe measured 22 mm. anteroposteriorly and 21 mm. vertically and horizontally. The cornea measured 10 by 10 mm. The lens and vitreous body appeared normal. After the globe was cut a small tumor-like elevation over the disk could be seen macroscopically. The nerve, including the tumor, was much enlarged throughout its entire length, the enlargement being greatest in the posterior half. Immediately behind the globe the nerve proper measured 2 mm. in diameter and gradually increased to 5 mm., tapering again toward the apex of the orbit. The surrounding tumor increased the size considerably, the transverse measurement over all being 5 mm. at the globe and 13 mm. at its greatest diameter. The nerve was eccentrically placed within the thickest portion of the tumor mass, the distention of the subdural space being more marked on one side than on the other. It was also apparently kinked. Some portions of the nerve showed little thickening of the sheath, though the nerve stem itself was more than twice its normal size. No part of the nerve or its sheath, however, was normal, so the growth must have extended back into the intracranial part of the nerve.

The globe was severed from the nerve, a short piece remaining attached. The nerve with surrounding tumor was cut in several pieces, and it and the globe were fixed in a 10 per cent dilution of formaldehyde U. S. P. Portions were later refixed in Zenker's solution.

Microscopic Examination.—Various stains were used in preparing the sections, including hematoxylin and eosin, Van Gieson's stain, Mallory's phosphotungstic acid and hematoxylin stain, Hortega's silver carbonate stain, Cajal's gold chloride stain and Weigert's stain.

Globe: The globe appeared slightly flattened in its anteroposterior diameter. The cornea and sclera appeared normal, though the latter was slightly thicker about the posterior pole. The scleral lamellae had become separated through shrinkage. The filtration angle was free and showed no evidence of inflammatory cellular change. Schlemm's canal was present and somewhat distended. The iris presented a varied picture, depending on the area studied. In some sections it appeared fairly normal, with only a slight increase in the normal cellular content. In other sections, evidently where the pupil was drawn to one side, there was marked thickening due to an enormous increase of large branching cells with large oval nuclei. Some of the cells contained pigment and showed all the characteristics of the ordinary chromatophores. Many were nonpigmented. Masses of pigment cells were scattered through the stroma. The clump cells were broken up, the pigment spreading into the stroma. There was a beginning ectropion of the uvea. The ciliary body was shrunken but otherwise appeared normal. The choroid was thin with the exception of an area on either side of the disk, where it was definitely thicker than normal. In this area it was heavily pigmented. There was an overgrowth of fibrous elements arranged in layers, with long spindle-shaped nuclei. Blood vessels in this area were fewer than normal, though this was evident throughout the choroid. This was probably due to shrinkage from the fixation in formaldehyde.

There was a definite elevation of the disk due to marked overgrowth of glial cells (fig. 10). This swelling consisted of a reticulated and markedly vacuolated tissue in which there were no connective tissue elements but those surrounding the blood vessels. The cells branched in every direction, the processes uniting and forming a vacuolated matrix. The glial nuclei were greatly increased in numbers. Some were large and oval and deeply stained; others were more elongated and spindle shaped. This glial proliferation extended back through the lamina cribrosa into the nerve proper. It also extended into the fiber layer of the retina in a

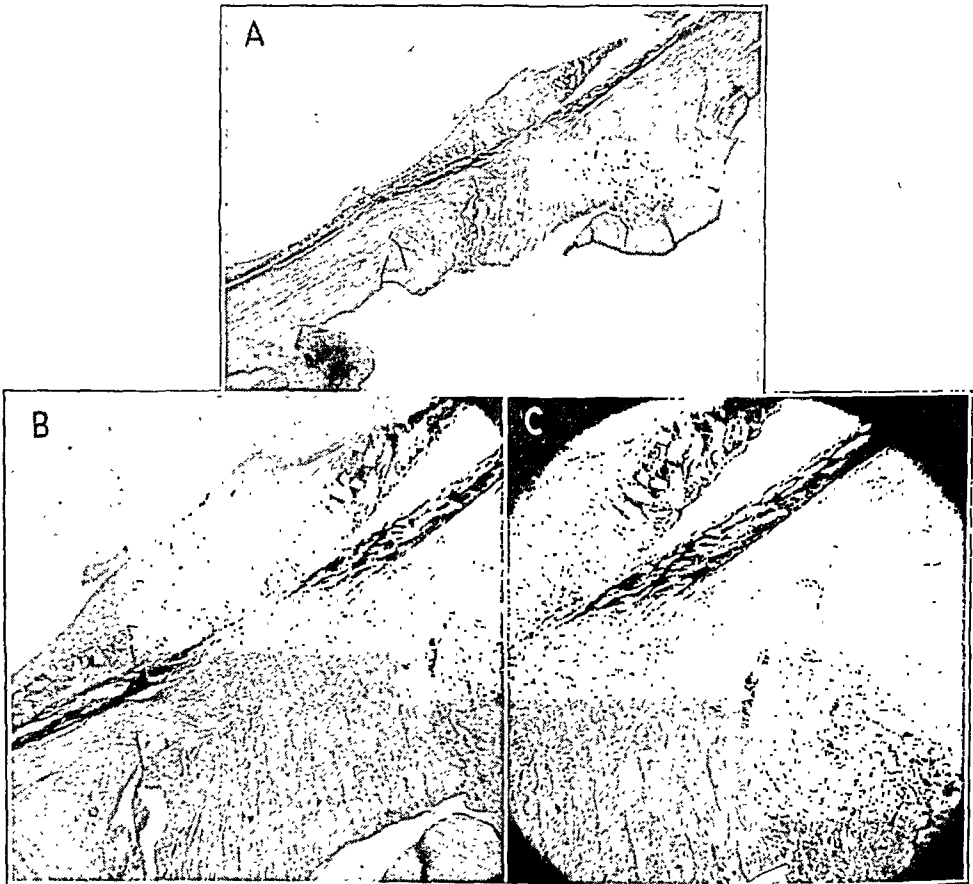


Fig. 10 (case 1).—Posterior segment of the globe. Note the glial proliferation of the nerve head, the cystic spaces in the retina and the proliferation of arachnoid cells at the anterior end of the vaginal space.

swollen area immediately surrounding the disk. The retina showed marked cystic degeneration beyond the borders of the swollen disk, the spaces being of considerable size in this location (fig. 10). For the most part they were within the nuclear layers. Farther out in the retina numerous small cystic spaces were seen situated in the outer nuclear layer as well. These cystic areas extended temporally, almost to the equator. Some contained a faintly stained transparent material and closely resembled ordinary cysts of the retina. Apparently these cysts had developed from stasis, as a result of interference with circulation brought

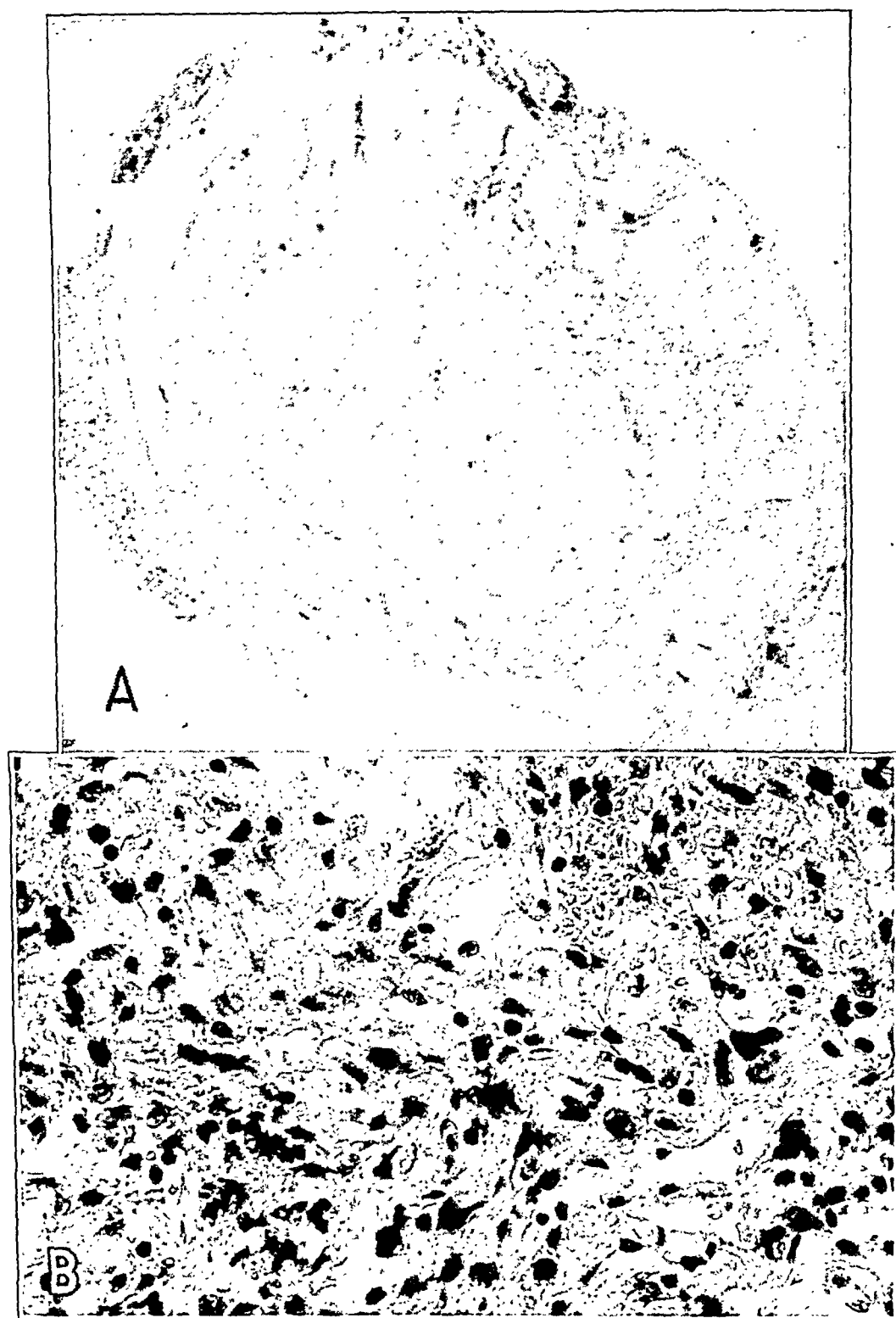


Fig. 11 (case 1).—*A*, section through the optic nerve and tumor. The section is taken near the globe. Note that the nerve stem is only moderately involved. The tumor involving the nerve sheath consists largely of proliferated arachnoid cells. *B*, arachnoid proliferation of the sheath adjoining the globe. (See figure 10.)

about by the tumor. The nerve fiber layer and ganglion cells were generally atrophic throughout the retina, with fine cystic spaces scattered through some of this area.

The lamina cribrosa was broken up by glial proliferation, though some connective tissue elements were still present. The optic nerve immediately behind the globe was of normal size, and its general appearance was fairly normal. There was, however, a definite increase in glial nuclei with some vacuoles. The pial sheath in this region was intact and of normal thickness. Outside this the subdural and arachnoid spaces were enormously distended (fig. 11 *A*) by an extremely cellular tissue, with short, oval, deeply stained nuclei which appeared much like

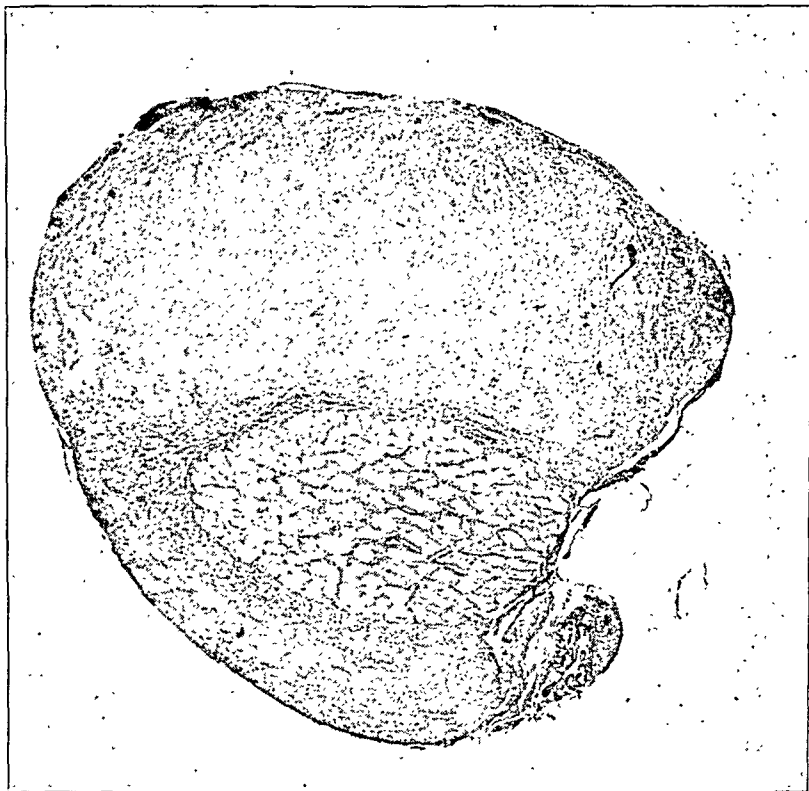


Fig. 12.—Horizontal section (oblique) of the optic nerve and tumor (stage 4). Note the large glial tumor surrounding the nerve stem.

abnormally proliferated arachnoid cells. These cells were in masses or sheets and showed no tendency to cluster formation or whorls (fig. 11 *B*). Some curled, wavy, pink-staining hyalinized bands of tissue coursed through the masses of cells, which appeared to be remnants of the arachnoid membrane. In this particular area glial fibers were not present, though transverse sections of the nerve a little farther back showed them in abundance.

Optic Nerve: The optic nerve, with the surrounding tumor, was cut transversely, and numerous sections were studied at different levels. The nerve evidently had a kink in it, since some sections were cut obliquely, while others showed double sections side by side, one being transverse and the other obliquely horizontal. Transverse sections nearest the globe showed the nerve stem within the pia to be normal in size (fig. 11 *A*). The connective tissue septums were somewhat thickened,

though their arrangement was like that of the normal nerve. The glial framework was accentuated and in most areas vacuolated. The nuclei of the glial cells were definitely increased in size and number. They were made up of large oval and round forms, the former predominating. Some were of slightly irregular oval contour. Many of the nuclei appeared in clumps, some of the cells being multinucleated. This appeared to be the result of proliferation or amitotic division. The nuclei showed a finely granular chromatin pattern, some having one large nucleolus and others two or more smaller dots. Vacuoles were especially prominent in the

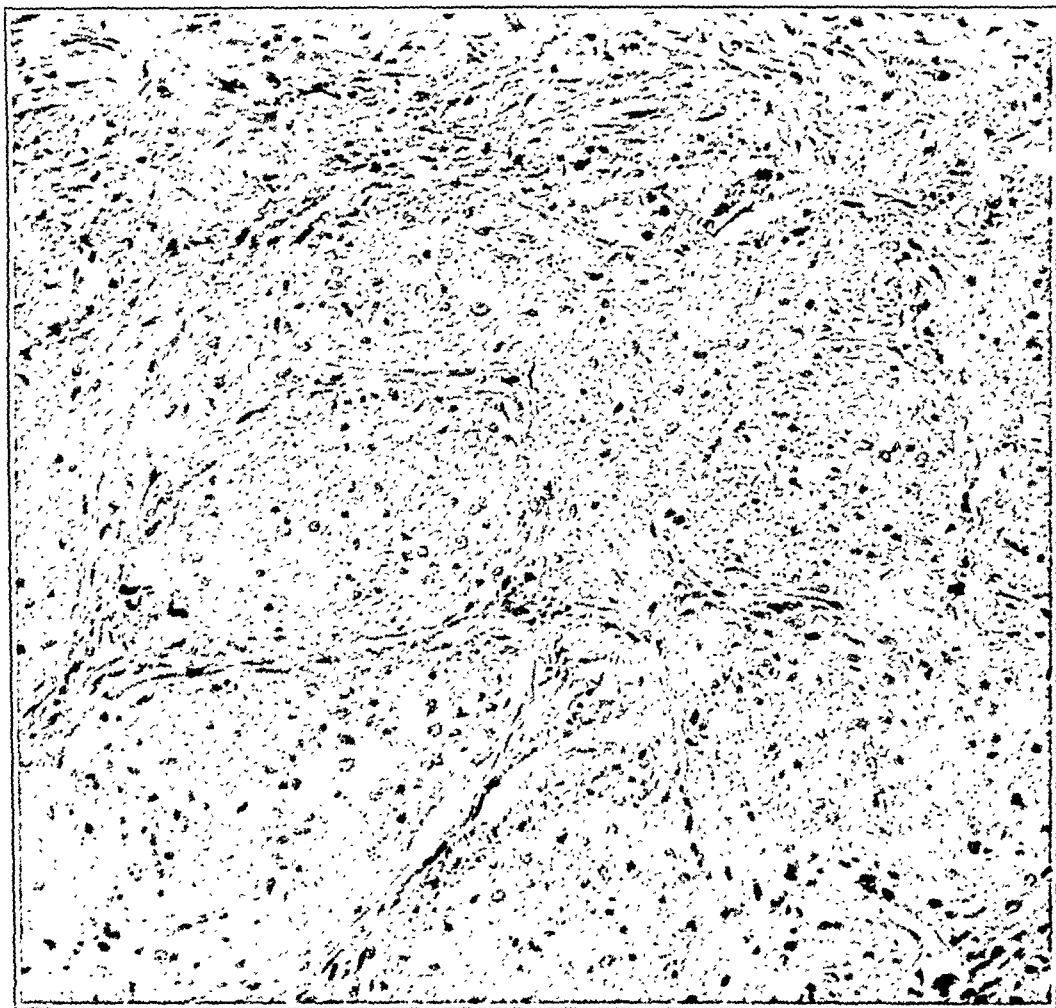


Fig. 13 (case 1).—Transverse section through the nerve stem (hematoxylin and eosin; $\times 200$). Note the increased glial nuclei. The tumor merges with a tumor in the nerve sheath (upper portion of illustration) which it has invaded.

cell body around the nucleus, toward the periphery of the nerve. The pial sheath was present though separated and somewhat broken up in places. Outside the nerve, within the pial lamellae, glial fibers were found which extended only a short distance. Beyond this there was an enormously thickened mass of arachnoid cells which distended the subdural space, as described in the portion of the nerve attached to the globe. The dura in this region was thickened. Farther back, in the thickest portion of the specimen, transverse sections showed the nerve cut obliquely (fig. 12). It was more than twice the normal size and was surrounded by a large glial tumor

mass. The nerve here showed the usual arrangement of the funiculi, separated by connective tissue septums. The nerve bundles were increased in size. Sections which were more exactly transverse showed a honeycombed, vacuolated pattern (figs. 13 and 14), with a marked increase in glial cells. Some of the vacuoles appeared to be holes or spaces where nerve fibers had disappeared, while others were in the cell body. The nerve fibers were degenerated, the myelin sheath failing to stain except in a few places.

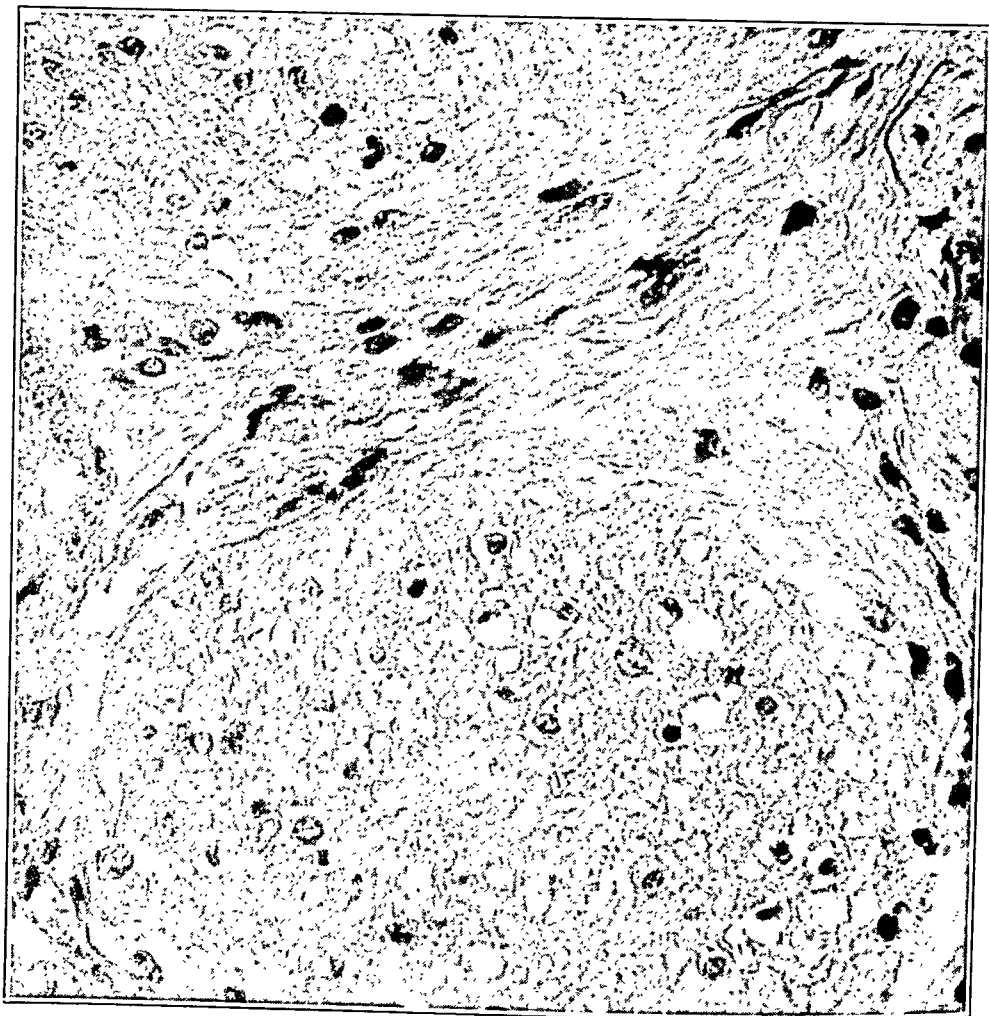


Fig. 14 (case 1).—High power magnification of the section in figure 13 (hematoxylin and eosin; $\times 550$). Note the glial cells, chiefly astrocytes (much enlarged); the large oval nuclei, and some oligodendrocytes. Note also the condensation of glial fibers bordering the fibrous septums, vacuolation of glial cells and the holes (sites of shrunken nerve fibers).

About the borders of the funiculi, adjacent to the connective tissue septums, there was a condensation of glial fibers which in places was dense and without vacuoles. This dense mass of fibers stained deep blue with Mallory's phosphotungstic acid-hematoxylin stain. The nuclei of the cells in this region were more elongated usually than those in the central portion of the nerve fiber bundles. The

connective tissue septums were intact but in places had been invaded by glial cells and masses of fibers. The pial sheath was broken up into lamellae, in some places being unrecognizable except for a few faintly stained pink strands. Glial

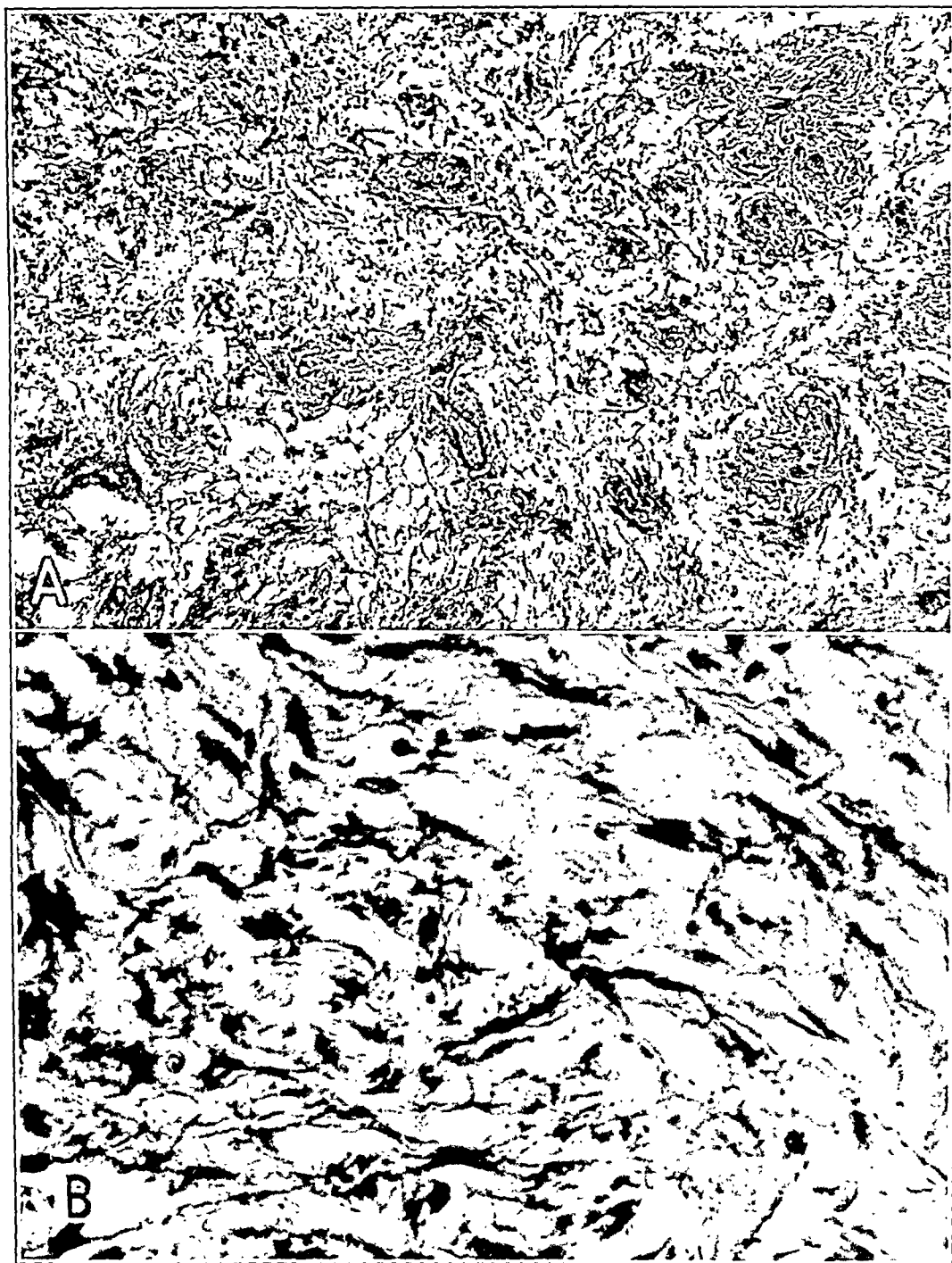


Fig. 15 (case 1).—*A*, section from the main body of the tumor in the nerve sheath—an astrocytoma (hematoxylin and eosin; $\times 150$). Note that the only connective tissue is that surrounding blood vessels. *B*, high power magnification of a section from the tumor in the nerve sheath, showing cells with branching processes (phosphotungstic acid stain; $\times 400$).

fibers, coarse and straight, penetrated the remains of the pia and were found in abundance throughout the tumor mass as far as the dural sheath, which they also invaded.

The tumor outside the nerve stem proper filled and distended the subdural space. It appeared to be made up largely of a reticulated mass of glial cells and fibers. The structure, however, was much more complex than that within the nerve. In some areas the growth was made up of a loosely meshed, highly vacuolated tissue (fig. 15). In some areas there were fairly large spaces, but no fluid or myxomatous material was found. The cells varied in contour, some being spindle shaped, with processes extending from one or both ends or more often branching in every direction. Small vacuoles were present within the cell body, many of them apparently fusing into larger vacuolated spaces. The tissue was more dense immediately beneath the dural sheath, where the cells in places were of a different type. These were evidently proliferating arachnoid cells. Neuroglial fibers permeated the entire tumor mass but were less abundant in the periphery. They were coarse and dense in places, with no definite cell attachments, while in other places they were finer and more wavy. They stained differentially with Mallory's phosphotungstic acid-hematoxylin stain in typical fashion, showing deep blue. Connective tissue elements were limited chiefly to isolated and broken strands of pial sheath and to that which surrounded the blood vessels. The latter were usually small and not a prominent feature of the tumor.

There was no evidence in the tissues of inflammatory involvement. No concretions or psammoma bodies were found. A few cytoid bodies were seen toward the outer border of the tumor mass near the dura. No part of the nerve studied was normal; so one would assume that the intracranial portion of the nerve was affected.

Differential staining with silver carbonate and gold chloride was unsatisfactory. Other stains, however, revealed beyond question the glial nature of the cells. Considering the marked similarity of this tumor to that in cases 2 and 3, the cells of which stained well with silver carbonate, the predominant cell type appeared to be astrocytes.

Diagnosis.—A diagnosis was made of Recklinghausen's disease of the abortive type, with glioma of the optic nerve stem (type, astrocytoma) invading the sheath.

Comment.—At the time of the original admission of the patient to the hospital little importance was attached to the pigmented spots in the skin. They were noted in the routine physical examination, but their significance was completely overlooked. The history of similar signs of Recklinghausen's disease in the mother and a younger brother was not obtained until fifteen months later, when the brother was admitted to the ophthalmic ward because of a slight unilateral exophthalmos. Similar café au lait-pigmented patches were then noted, and later more intensive study was undertaken. The family had attached no importance to the spots, the mother, when questioned, referring to them as "liver spots." The patient's twin brother was thoroughly examined and found to be normal. He was free of pigmented patches, and his vision and fundi were normal; roentgenograms showed his skull, including the optic foramens, to be normal.

An interesting feature of this case was the presence of glial proliferation of the nerve head, with large cystic spaces in the retina adjacent to the disk, similar to that noted in Verhoeff's case. This feature was recognized before operation and described clinically as a low detachment of the retina, with probable invasion of the retina by a tumor.

The section of nerve attached to the eyeball showed unusual proliferation of arachnoid cells. These diffuse masses of cells in some ways resembled the meningiotheliomatous type of meningeal tumors described by Bailey and Bucy. They did not, however, have the characteristics of the ordinary endothelioma or meningioma of the optic nerve, since no whorls or nests of cells and no psammoma bodies were present. Verhoeff discussed this feature of the tumors in the cases he reported, and in a personal communication he stated that he considered "the cell masses were merely proliferated arachnoidal cells and not a true endothelioma."

The marked thickening of the iris on one side and the thickened choroid presented an appearance suggesting early neurofibromatosis. The histologic picture, however, was by no means typical, nor were there other changes in the globe usually associated with this condition. The irregularity of the pupil, noted on clinical examination of the eye, was due to this localized thickening of the iris. It was much more advanced in case 3, in which the growth proved to be typical neurofibromatosis of the ciliary nerves in the iris.

The invasion of the nerve sheath by the tumor was much farther advanced in this case than that in case 2. The pia and arachnoid were almost completely destroyed in places. Only isolated remnants of the pia remained in some areas, while in others small masses of arachnoid cells were the only indication of the arachnoid membrane.

The dura over the thickest part of the tumor was invaded and almost destroyed by the growth in some places.

This patient has been examined at least once a year since operation, and there has been no change worthy of note. In a recent examination she appeared to be in good health. The pigmented patches in the skin had definitely increased in size, but no other peripheral changes had developed. There was no evidence of local recurrence in the orbit. Roentgenograms of the optic foramen showed some enlargement, but the reading was unsatisfactory, due, possibly, to the distortion of the orbit from the Krönlein operation or to erosion of the canal from further progress of the growth.

From the appearance of the sections of the tumor near the apex of the orbit, further growth of the tumor within the intracranial portion of the nerve is to be expected. If this is occurring, its progress must be exceedingly slow, for nearly eight years have elapsed since the operation.

Exenteration of the orbit in this case was unnecessary, as will be noted in the 2 succeeding case reports. The relatively benign nature of the tumor was unknown at the time of operation, and a malignant growth was suspected.

CASE 2.—*Diagnosis of primary tumor of the optic nerve; retrobulbar removal of the optic nerve with the tumor; preservation of the globe; glioma of the optic nerve, invading the sheath, and Recklinghausen's phenomena—cutaneous café au lait-*

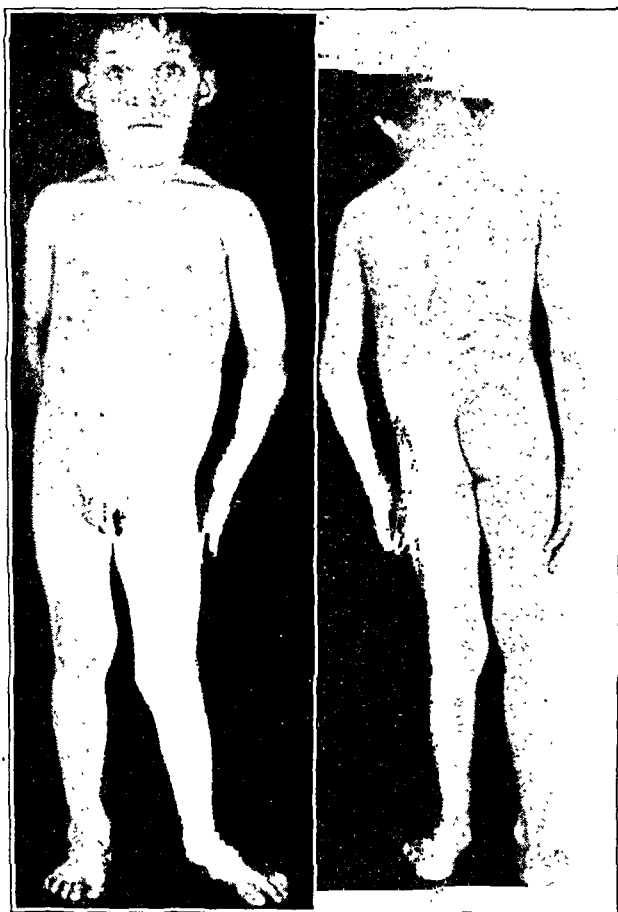


Fig. 16 (case 2).—A. H., aged 4 years, with a glioma of the optic nerve (left eye). The photographs were taken one year before the patient's admission to the ophthalmic ward. Note the café au lait-pigmented spots, scoliosis, deformity of the left leg and slight widening of the left palpebral fissure.

pigmented spots, scoliosis and congenital deformity of the fibula; no recurrence; period of observation, six years (figs. 16 and 17).

A. H., aged 6 years, a brother of the patient described in case 1, was first admitted to the orthopedic department of the Wisconsin General Hospital on Oct. 16, 1931, the same day that the sister entered the ophthalmic ward. At this time, however, there was no complaint concerning his eyes, and he was admitted for a correction of a deformity of the left leg, which proved to be a congenital

absence of the fibula. There was also scoliosis of the spine. Bone grafts were made, and the child was readmitted to the hospital six months later for further grafts. He was admitted a third time on Jan. 5, 1933, and while in the hospital on this occasion he complained of pain in his left eye and stated that he could not see with it. He was then referred to the ophthalmic clinic, where a slight exophthalmos of the left eye was noted, measuring 4 mm. with the exophthalmometer (fig. 18). The eye projected directly forward, and there was a marked limitation of upward rotation. Movements in all other fields were normal. The

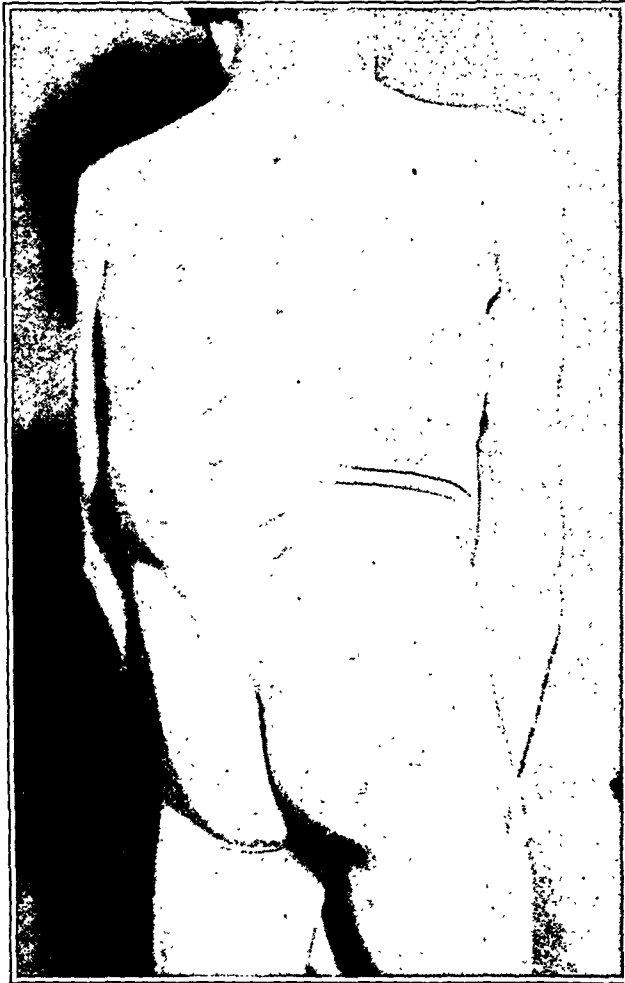


Fig. 17 (case 2).—Appearance of the patient five years after operation. Note the scoliosis and café au lait-pigmented patches in the skin.

pupils were equal and reacted normally to light and in accommodation. The media were clear. Examination of the fundus revealed an elevation of the disk of 2 diopters, with marked blurring of the margins of the disk. There were no hemorrhages or exudates. Vision in the right eye was 20/30 and in the left eye 20/70.

A diagnosis of tumor of the optic nerve was made and operation advised. The child was observed for two months because orthopedic work on the leg required further attention. During this time the choking of the disk increased somewhat, but there was no change in the exophthalmos. The vision decreased to 20/100.

The right eye was normal.

Previous Medical and Family History.—The child was born at full term, but delivery was difficult, the mother being in labor twenty-four hours. No instruments were used. He had pneumonia at the age of 4 months. The boy had been in the orthopedic ward off and on for a year and a half for correction of a

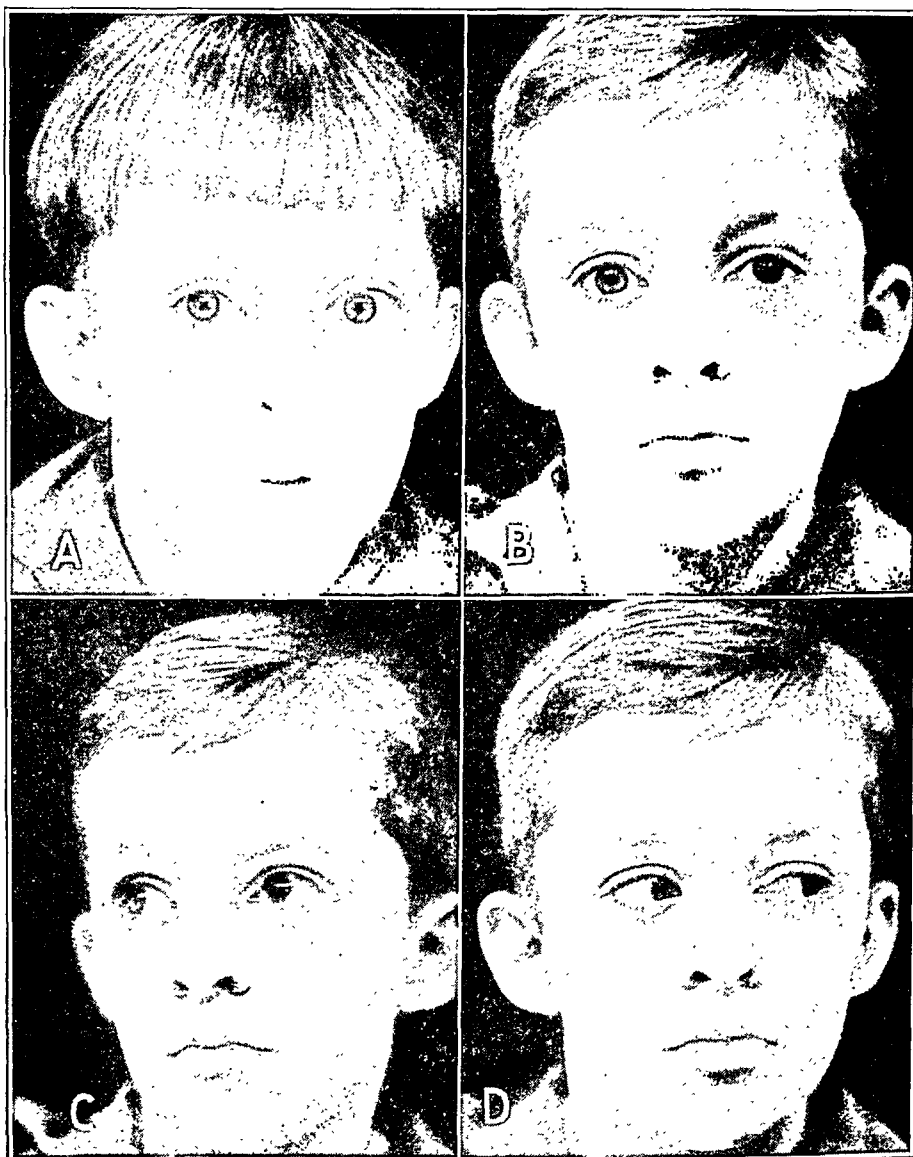


Fig. 18 (case 2).—A. H. aged 5 years. *A* shows slight exophthalmos of the left eye (4 to 5 mm.). *B* shows the patient four months after operation. Retrobulbar removal of the entire tumor of the optic nerve was done. Note the faint line of scar along the lower orbital rim. *C* and *D* show the appearance of the eyes when the patient looked to the right and left. Note the perfect motility and position of the globe.

congenital absence of the lower end of the fibula, with talipes valgus and marked bowing of the tibia, as stated previously. The sister had a tumor of the optic

nerve, as recorded in case 1. The mother had Recklinghausen's disease in a mild form, the manifestation consisting of numerous café au lait-pigmented patches on the skin of the trunk and extremities and several soft, elevated mollusca fibrosa on the abdomen, back and arm and a number of firm, knoblike nodules embedded in the skin of the chin.

General Examination.—Aside from postural deformity produced by a congenital defect, the only general findings of interest were multiple smooth light brownish patches on the skin of the back, abdomen, thigh and buttocks, similar to those noted on the patient's sister. These were 1 to 2 cm. in diameter, were not elevated and did not blanch on pressure. All general laboratory tests gave negative results, and the Wassermann reaction was negative. Roentgenograms of the skull, including lateral, stereoscopic and anteroposterior flat views, showed the cranial bones to be thin, but there was no increase in convolucional markings and no spreading of the sutures. The sella turcica was normal in size, and the clinoid processes showed no evidence of erosion.

Operation (March 27, 1933).—Since operation on the sister of this child had revealed a tumor of the optic nerve, apparently relatively benign, I decided in this instance to remove the optic nerve through the soft tissues, thus preserving the globe. Further, I wished to avoid the deformity incident to the Krönlein operation or to evisceration.

With the patient under anesthesia produced by avertin with amylene hydrate supplemented by nitrogen monoxide, a curvilinear incision was made through the skin along the lower rim of the orbit and carried down to the bone (fig. 19). The periosteum was incised and elevated along the floor of the orbit as far back toward the apex as possible. A linear incision was then made through the periorbita, and the fat and muscles were pushed aside with narrow gauze packing. The nerve was easily isolated and was found to be uniformly enlarged throughout its entire length, being about 10 mm. in diameter. The nerve was carefully isolated and tied with chromic catgut at the apex of the orbit as far as one could reach. It was similarly tied near the attachment to the globe and then excised. The wound was closed, deep catgut sutures being used in the periosteum and silk sutures in the skin.

Postoperative healing was attended by considerable reaction, with swelling of the lids and proptosis of the globe. A neurotrophic opacity developed in the lower central part of the cornea on the eighth day but healed without breaking down, though a permanent light nebula of the cornea remained. The cornea was anesthetic, and it had to be protected for some time to prevent drying. The general reaction and exophthalmos finally completely subsided, with return of full motility of the globe and luster to the cornea. The eyes remained perfectly straight until recently, when a slight divergent squint was noticed. There was no exophthalmos or enophthalmos. The left pupil remained dilated to about 5 mm. The corneal anesthesia has persisted. The palpebral fissure remained slightly wider than that of the right eye.

Examination of the Fundus (postoperative, on the operating table).—The fundus of the eye was examined at the end of the operation, and an increased congestion of the nerve head was the only change that could be noted. A note on the sixth postoperative day stated that the pupil was semidilated and fixed; the fundus was as described postoperatively; the vessels were full and of normal caliber; and the color of the nerve head had not changed.

Two years after the operation (Feb. 8, 1935) the boy returned for observation, at which time the following notes were made: Vision was 20/20 in the right eye

and nil in the left eye. The right eye was normal. The position of the left eye was normal, with slight widening of the palpebral fissure. The extraocular movements of this eye were normal. There was a slight limitation of the levator muscle. The cornea showed infiltration of stroma on the temporal side just below the center, which extended to the limbus; this was faint, however, and scarcely noticeable. The anterior chamber was of normal depth. The pupil was slightly larger than that of the fellow eye; it dilated with atropine but did not react to light. The media were clear. The disk showed total atrophy of the nerve, with sharp borders. The vessels were slightly constricted, though they contained blood. The light streak was poorly seen in both arteries and veins. It was definitely present in the larger vessels near the disk but was less distinct in the remainder

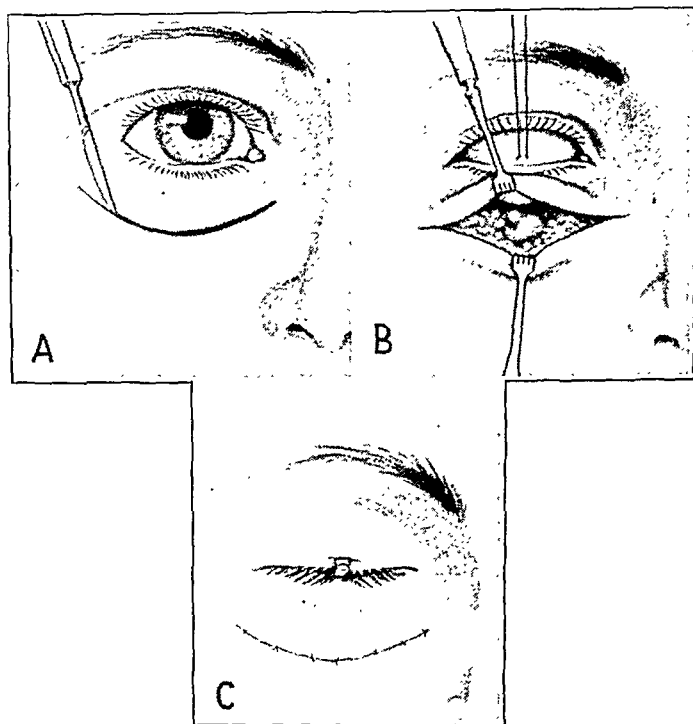


Fig. 19.—Operative procedure used for retrobulbar removal of a tumor with preservation of the globe. An incision is made through the skin along the lower margin of the orbit, as in *A*. The enlarged optic nerve is exposed, as in *B*. The nerve is tied off at the globe and the apex of the orbit, after which it is excised. The inferior rectus muscle may be detached and resutured, but no detachment of muscles was used in cases 2 or 3. The lid is sutured to prevent possible damage to the cornea, as in *C*. The wound in the skin is closed with silk sutures.

of the vessels in the periphery. There were no hemorrhages or exudates and no pigmentation.

Three and one-half years after operation (Aug. 6, 1936) the boy was again seen, and the following observations were recorded: The left eye was quiet and white. The position of the eye was good (fig. 18 *B*, *C* and *D*). The extraocular movements were normal and free in all fields. There was a slight divergence of

5 degrees. Convergence power was present but poor. The cornea showed a faint nebula 1.5 mm. wide, irregular in outline and extending across the inferior half of the cornea from the temporal side a little beyond the center. The pupil was about 5 mm. in diameter. There was anesthesia of the cornea. The media were clear. The nerve head was oval and perfectly flat and white, and the margins were sharply defined. The vessels were slightly smaller than normal; the arteries were filled. Some pigmentary changes were seen in the periphery of the fundus. Examination on Aug. 15, 1938, showed the left palpebral fissure to be slightly more narrow than the right. The left globe was slightly less prominent than the fellow eye. There was a divergence of 5 degrees. The motility was perfect in all directions of gaze. The cornea was smooth and glistening, with a faint nebula extending horizontally across it from the temporal border to the midline. The remainder of the cornea was clear. The pupil dilated well with homatropine hydrobromide; the media were clear, and the

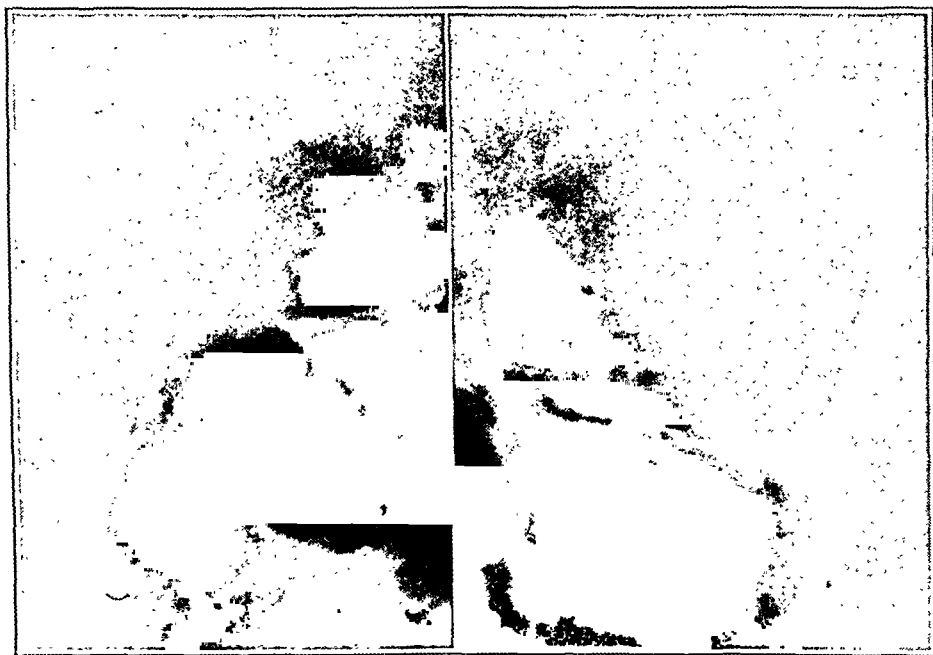


Fig. 20 (case 2).—Roentgenograms of the optic foramens (actual size). Both foramens are slightly enlarged, and the left is bean shaped.

crystalline lens and vitreous appeared normal. The disk was oval, flat and white, and the margins were sharply defined. The larger vessels showed a slight reflex as they left the disk, more definite in the arteries, and the light streak was soon lost as the vessels advanced toward the periphery of the fundus. The fundus had a normal reddish glow, and there was no abnormal pigmentation except for a few spots far out in the periphery. A photograph of the fundus showed no change from that taken two years earlier.

The right eye appeared normal; the fields and blindspots were normal.

A report of roentgen examinations follows: The skull appeared to be within normal limits; the sella turcica was normal in contour. The optic foramens measured: 5 by 5 mm., the left being slightly bean shaped (fig. 20). Further study of the films (lateral stereoscopic views), however, revealed a thin necklike shadow extending under the anterior clinoid process from the upper anterior edge

of the body of the sella turcica. This shadow was not so definite as those observed in cases 1 and 3. It was not present in the original films, taken five years earlier.²

Macroscopic Examination.—The excised portion of the nerve surrounded by the tumor measured 24 mm. in length. It was slightly oval, measuring 8 by 9 mm. (fixed specimen) in its thickest portion and tapering slightly toward each end, being 4 mm. at the globe and 5 mm. at the apex of the orbit. The dural sheath was intact throughout the length of the nerve. It was perfectly smooth, without adhesions to surrounding structures. The nerve stem within the pia was enlarged and was surrounded by a subdural tumor mass which was slightly thicker on one side. The thicker side measured 2 mm.; the thinner, 1 mm. The nerve stem proper, without the tumor in the sheath, measured 5 by 7 mm. in the thickest portion.

Microscopic Examination.—The nerve, cut into pieces, was fixed in a 10 per cent dilution of formaldehyde U. S. P. and in Zenker's solution and embedded in paraffin. Transverse sections were made at different levels throughout its entire length. The sections were stained with hematoxylin and eosin, van Gieson's stain, Mallory's phosphotungstic acid-hematoxylin stain, Hortege's silver carbonate stain for astrocytes and oligodendrocytes, the Weil-Davenport ammoniacal silver stain, Weigert's stain, Weil's iron hematoxylin and others.

Sections of the tumor showed considerable variations, depending on the level at which the specimen was cut. Sections from the thickest portion of the tumor revealed a fairly definite pattern (fig. 21). The nerve stem was much enlarged due to abnormal proliferation of the glial elements. A fairly thick tumor mass surrounded it, which was also glial in nature. It apparently arose from the tumor cells within the nerve which had invaded the pia and extended into the subarachnoid space. Surrounding this was the arachnoid sheath, much thickened, the outer cells of which showed marked proliferation. Surrounding all was the dural sheath.

Thus the tumor mass might be roughly divided into five layers or zones: first, the tumor of the nerve stem; second, the pial sheath broken up and invaded by glial cells and fibers; third, the tumor in the nerve sheath, gliomatous in nature; fourth, the arachnoid sheath, much thickened from proliferation of the arachnoid cells, and, fifth, the dural sheath.

Detailed Study of Transverse Sections: The nerve stem was more than three times its normal size. The arrangement of the funiculi, or bundles of nerve fibers, was preserved as in the normal nerve, though the bundles were considerably enlarged. The fibrous framework, or septums, showed the usual normal arrangement, but they were thickened and, in places, somewhat distended. The septums throughout the nerve were clearly demarcated, but there was no active proliferation of connective tissue cells in the septal wall. Small blood vessels

2. Since this paper was written, the patient has returned for examination. Definite evidence of an early glioma of the right optic nerve has developed since the last study (one year ago), consisting of slight exophthalmos, early choking of the disk and reduced vision. There is no evidence of recurrence of the growth in the left orbit. There are also signs of intracranial involvement. This indicates the lesions are probably multiple and that the present condition represents a further development of areas of abnormal glial proliferation in the right optic nerve and in the brain and is not necessarily an extension from the original tumor in the left nerve.

and, in places, some loosely arranged cells with oval nuclei were found within the intraseptal spaces, and glial fibers (stained blue with phosphotungstic acid stain) were also encountered within some of these spaces. The funiculi were transformed into a meshwork of honeycombed spaces, studded with innumerable glial cells (fig. 22). The honeycombed spaces were of irregular size, some of which appeared to be empty holes, the site of nerve fibers which had either disappeared or shrunk. The meshwork was evidently the glial framework, somewhat thickened and therefore more prominent. Some of these holes may have been due to shrinkage from the fixation or embedding in paraffin. (Similar holes are frequently seen to a less degree in otherwise normal nerves, and I have often observed them in degenerating nerves.) Many of these spaces, however, were vacuoles in the glial cells. With hematoxylin and eosin and Van Gieson's stain

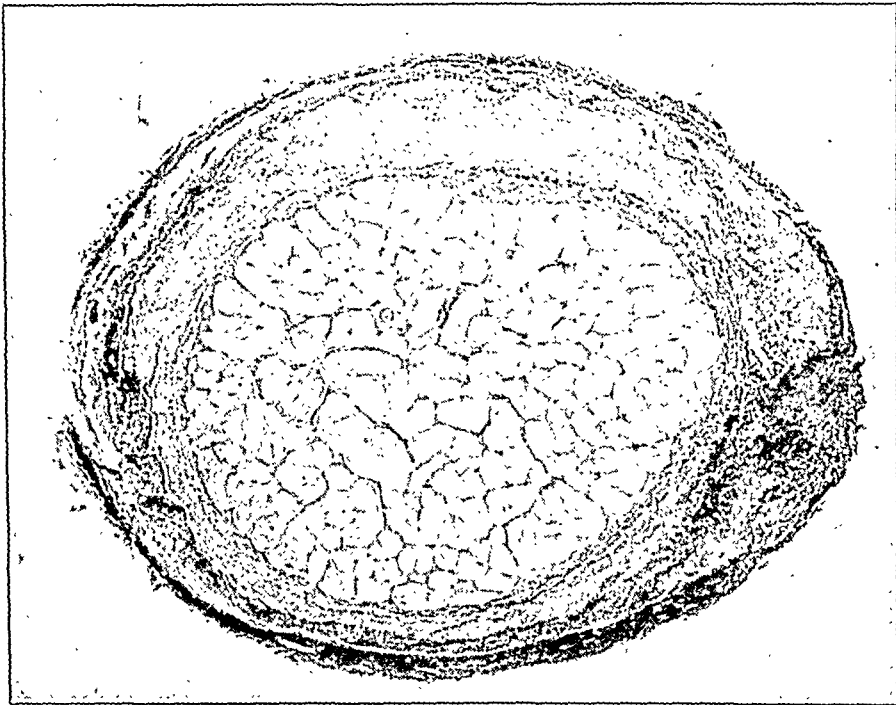


Fig. 21 (case 2).—Glioma of the optic nerve. Transverse section of the nerve and the surrounding tumor (stage 3; about $\times 20$). The nerve stem is much enlarged. The glial tumor has penetrated the pia and involves the sheath as well as the nerve stem. Note the thin layer of arachnoid cells just beneath the dural sheath.

the vacuoles were seen adjoining the nuclei. With silver impregnations the cell body was often clearly outlined, but vacuoles did not always appear definitely within them. The nuclei of the glial cells were seen most clearly with hematoxylin and eosin and Van Gieson's stain. They appeared to be of two principal varieties, large oval and round forms. Some were smaller and round. In places irregularly shaped oval forms were seen. Chromatin figures were fairly prominent, and one or two large dots were found in some of the nuclei. The nuclei were more irregular in shape and much larger than those of the normal nerve, which they otherwise resembled (fig. 23). They were especially hyperchromatic at either end of the nerve, where glial proliferation appeared in an early stage, while fiber formation was not so advanced. Multinucleated cells were frequently seen, grouped in masses, but no mitotic figures were found. The cell bodies were

so distinct and fibrous expansions so numerous and coarse that they were clearly brought out with phosphotungstic acid stains (fig. 24 *A*). With Hortega's silver carbonate stain for astrocytes and oligodendrocytes and the Weil-Davenport ammoniacal silver stain they were well impregnated and could therefore be identified as astrocytes and oligodendrocytes.

By these staining methods most of the cells which were scattered through the funiculi were found to be astrocytes. They had large oval translucent nuclei.

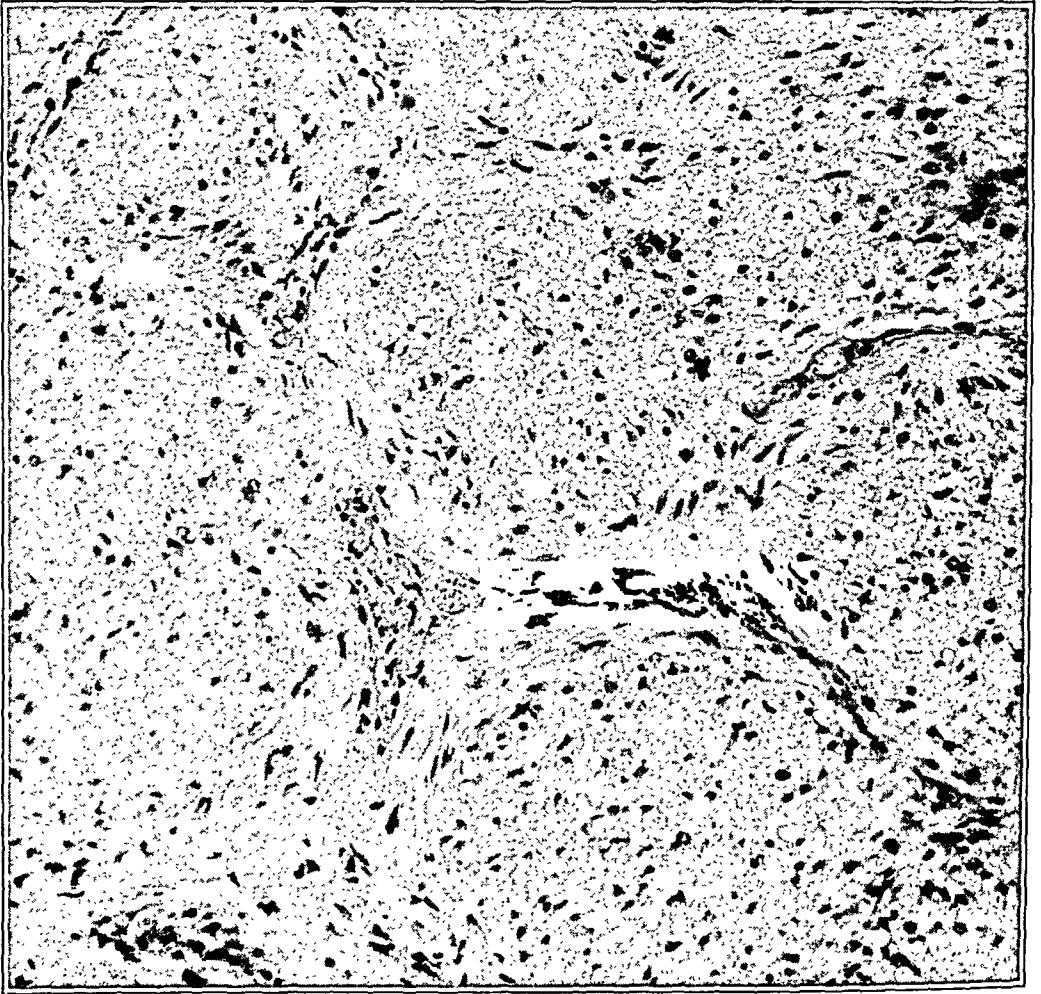


Fig. 22 (case 2).—Transverse section through the nerve stem and the main body of the tumor (hematoxylin and eosin; $\times 200$). Note the marked increase in glial cells with condensation of glial fibers bordering the fibrous septums.

Each nucleus was surrounded by a cell body, irregular in shape, the cytoplasm of which appeared granular (figs. 25, 26, 27 and 28). Extending out from this irregularly shaped cell body were numerous processes branching in every direction. These were dense, coarse and wavy and could be followed for long distances. These astrocytes were of the fibrillary type. Masses of cells, often arranged in rows, were seen, with long dense fibers streaming in every direction. Frequently, however, they assumed a parallel arrangement, especially when passing

from one funiculus to another (figs. 27 and 28). Where a funiculus was completely surrounded by fibrous septums, the glial fibers collected in a concentric, laminated, feltlike layer bordering the septums. Here the nuclei of the glial cells became more elongated and lost the typical shape which they assumed in the more loosely honeycombed network of the central part (figs. 22 and 23). Oligodendrocytes of fairly typical contour were scattered about in the funiculi (figs. 29 and 30). They usually had a round or slightly oval nucleus, which was

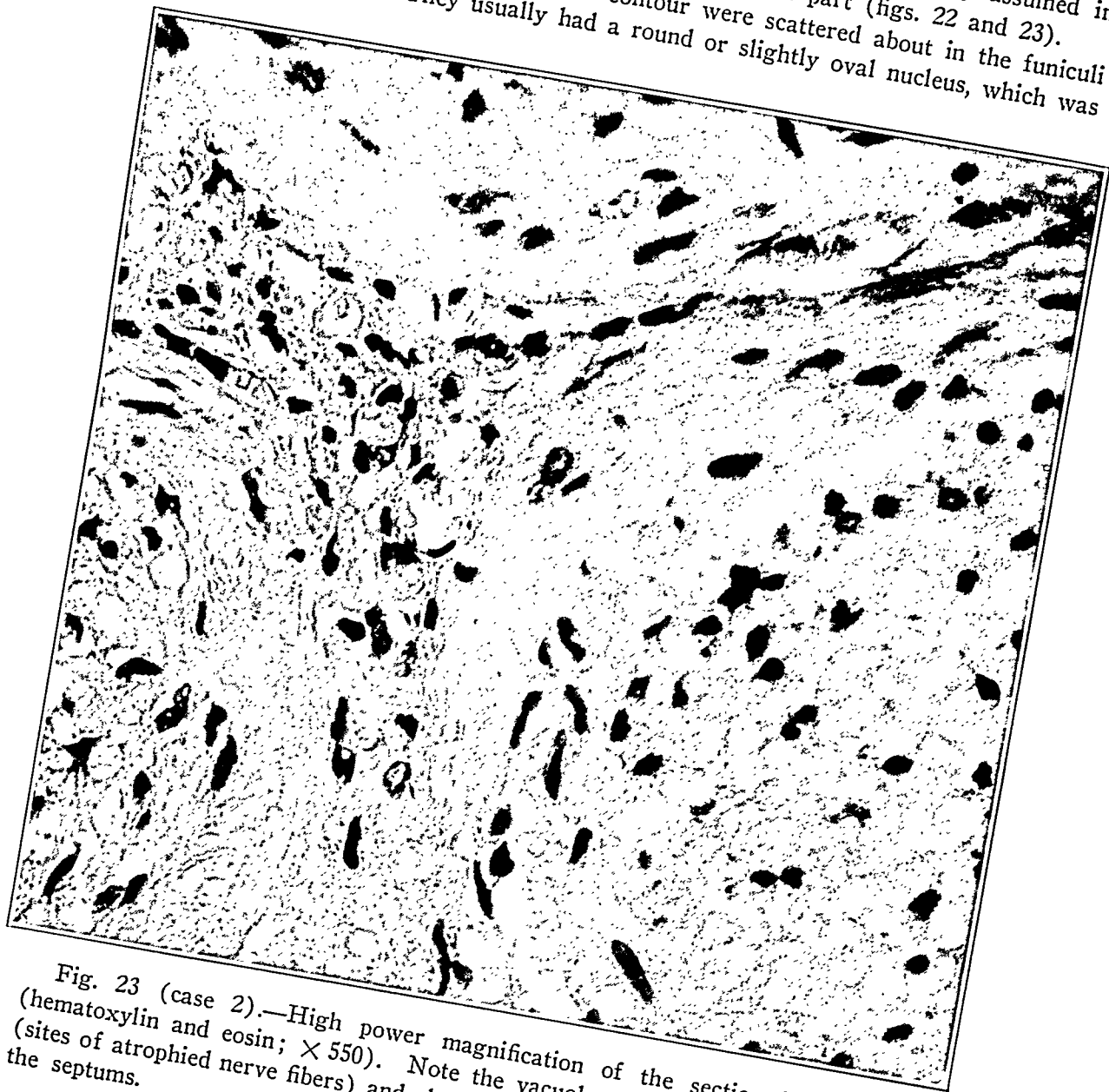


Fig. 23 (case 2).—High power magnification of the section in figure 22 (hematoxylin and eosin; $\times 550$). Note the vacuoles in glial cells and the holes (sites of atrophied nerve fibers) and also the dense masses of glial fibers bordering the septums.

often translucent, with a small cell body containing densely impregnated granules. Short curvy processes were seen extending out from the cell body for a short distance.

Subseptal attachments were clearly brought out by special stains, some of the processes being quite coarse (fig. 26). These could be seen extending from rows of astrocytes which bordered the felted bands of glial fibers. These subseptal feet

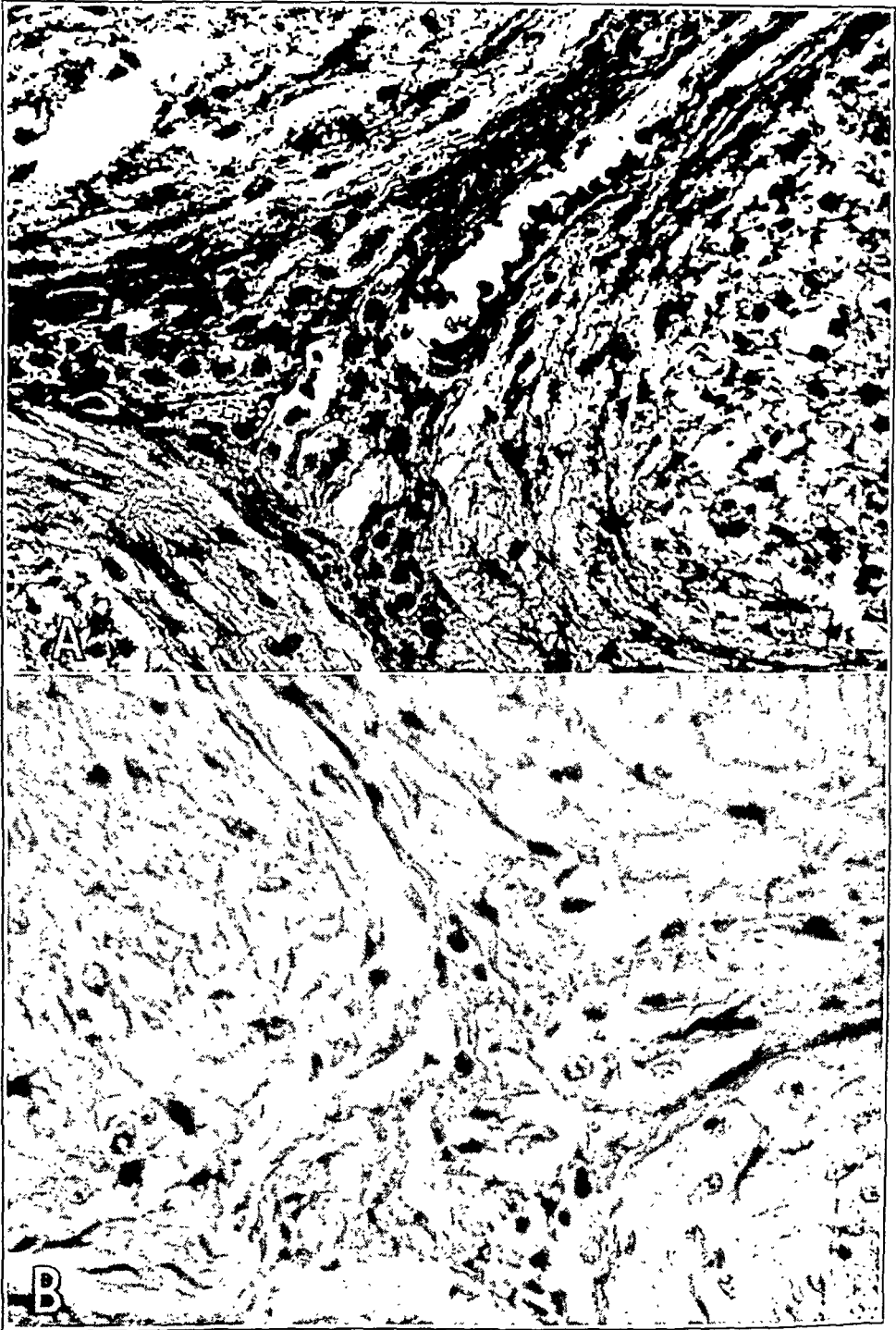


Fig. 24 (case 2).—*A*, high power magnification of the section in figure 22 (phosphotungstic acid stain; $\times 400$). Three funiculi are seen abutting a fibrous septal space. Note the branching process of cells. *B*, same section as in *A* (hematoxylin and eosin; $\times 500$).

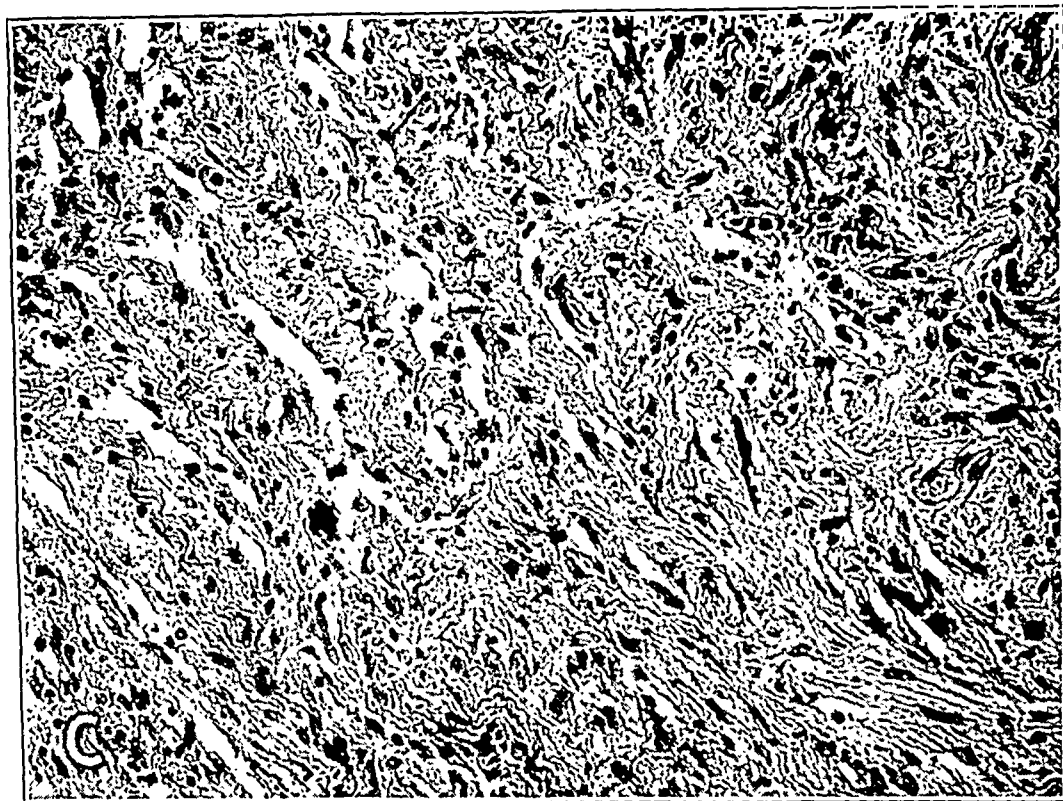


Fig. 24 (case 2).—C, detail of the tumor of the nerve sheath in another area; $\times 200$. Note the coarse glial fibers and cytooid bodies. (From the section shown in figure 21.)

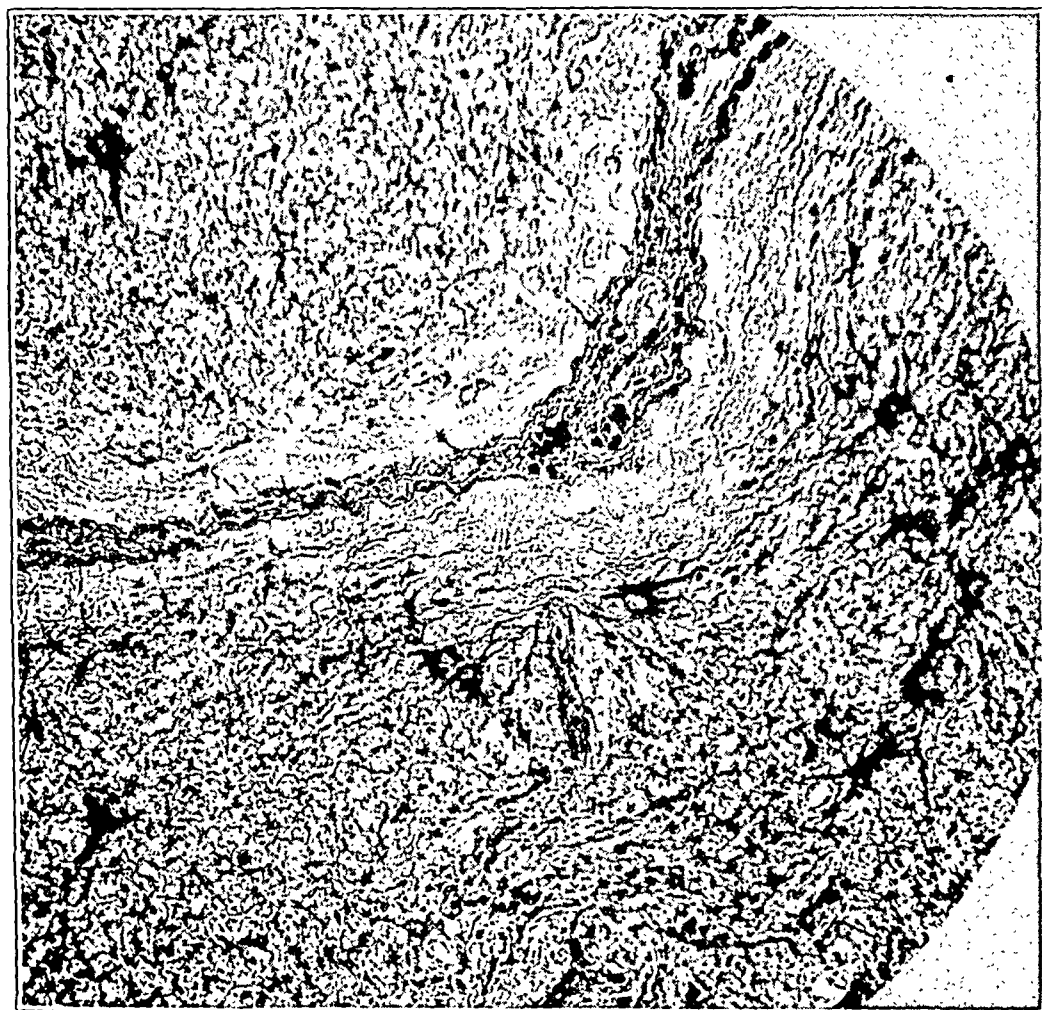


Fig. 25 (case 2).—Section from the tumor (Weil-Davenport stain; $\times 400$). Fibrous septums are seen surrounded by dense glial fibers. Note the astrocytes.

extended at right angles through the felted glial masses to reach the fibrous septums. Subpial attachments were not so definite, though they were seen at times. The condensation of glial fibers (figs. 22, 23, 25 and 26) bordering the connective tissue septums was a striking feature of sections from the nerve in this case as well as in cases 1 and 3. These felted bands, which were composed of masses of glial fibers, were not so definite about the funiculi which abutted the pial sheath. They were present in some places to a slight degree, but they

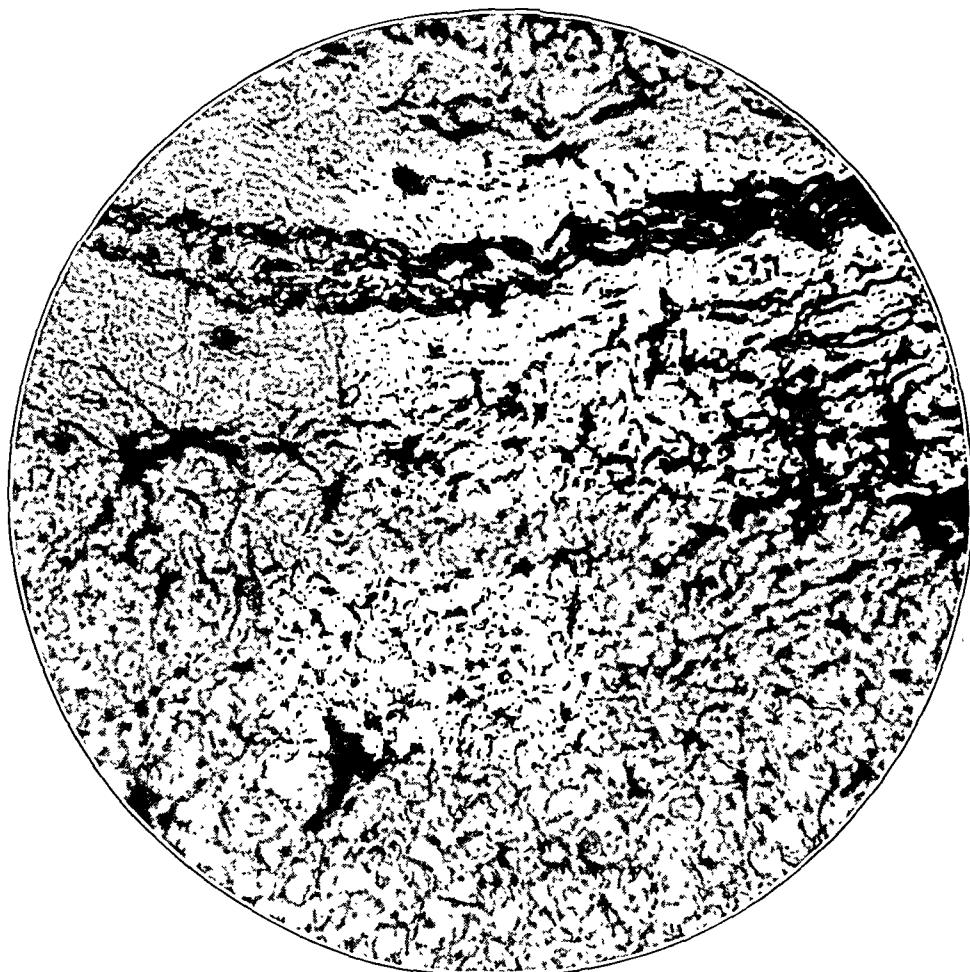


Fig. 26 (case 2).—High power magnification of section shown in figure 25. Note the large fibrous astrocytes bordering septums and subseptal expansions.

were conspicuously absent in most areas (fig. 31). Apparently the glial fibers had penetrated the pia in many places; so this particular arrangement or formation was lacking. Myelin sheaths were present in some places, but in most areas they failed to stain.

After the glial fibers left the nerve stem by penetration of the pial sheath in innumerable places, they encircled the nerve in transverse sections, running between and breaking up the pia into lamellae. The fibers appeared coarser and more dense when they were viewed in transverse sections (fig. 31).

Remnants of the pial sheath were present in most sections, but frequently they consisted only of isolated strands of pink-stained, wavy, hyalin-like tissue, embedded in glial fibers, which stood out in sharp contrast since they stained deep blue. Where the tumor was not so far advanced, the pial sheath area was much wider, though the lamellae were separated and distended by gliomatous tissue. The glial cells and fibers extended out beyond the pial zone, where they formed the main body of the tumor of the sheath, distending the subarachnoid space (fig. 32). The tumor here consisted of a reticulated mass of tissue, fairly dense in

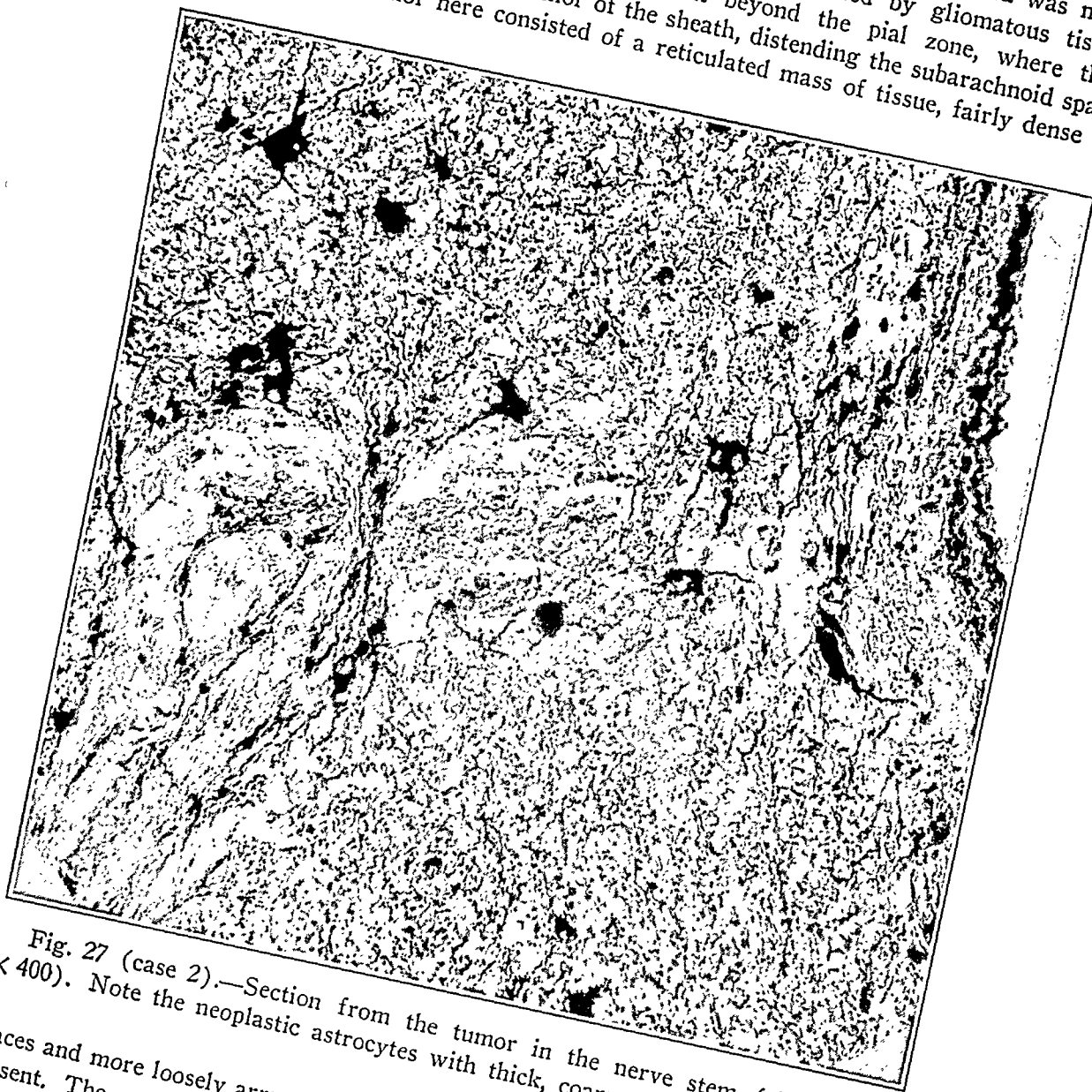


Fig. 27 (case 2).—Section from the tumor in the nerve stem (silver stain; $\times 400$). Note the neoplastic astrocytes with thick, coarse processes.

places and more loosely arranged in others. Many vacuoles and larger spaces were present. The vacuoles were in the cell body, while the larger spaces appeared to arise from a breaking down or fusion of the vacuoles. No myxomatous material was found in these spaces. The cell bodies were more spindle shaped in this region, long processes extending from each end (similar to fig. 15 B). They resembled the cells of the densely felted areas of the nerve stem. Typical star-shaped cells, however, were occasionally seen, with coarse wavy processes extending in every direction. The cells apparently communicated with each other

in some areas and therefore might be described as a "coarsely reticulated syncytium." The outer aspect of the gliomatous mass of tissue was bordered by the arachnoid sheath and the dura.

The arachnoid consisted of hyalin-like, wavy, strands of tissue, staining deep pink (phosphotungstic acid), embedded in and on the outer surface of which were masses of proliferating arachnoid cells. This area also differed from that in case 1 in that the glial cells and fibers had not penetrated or invaded it to such a marked degree. Outside this thickened arachnoid the dura was present throughout

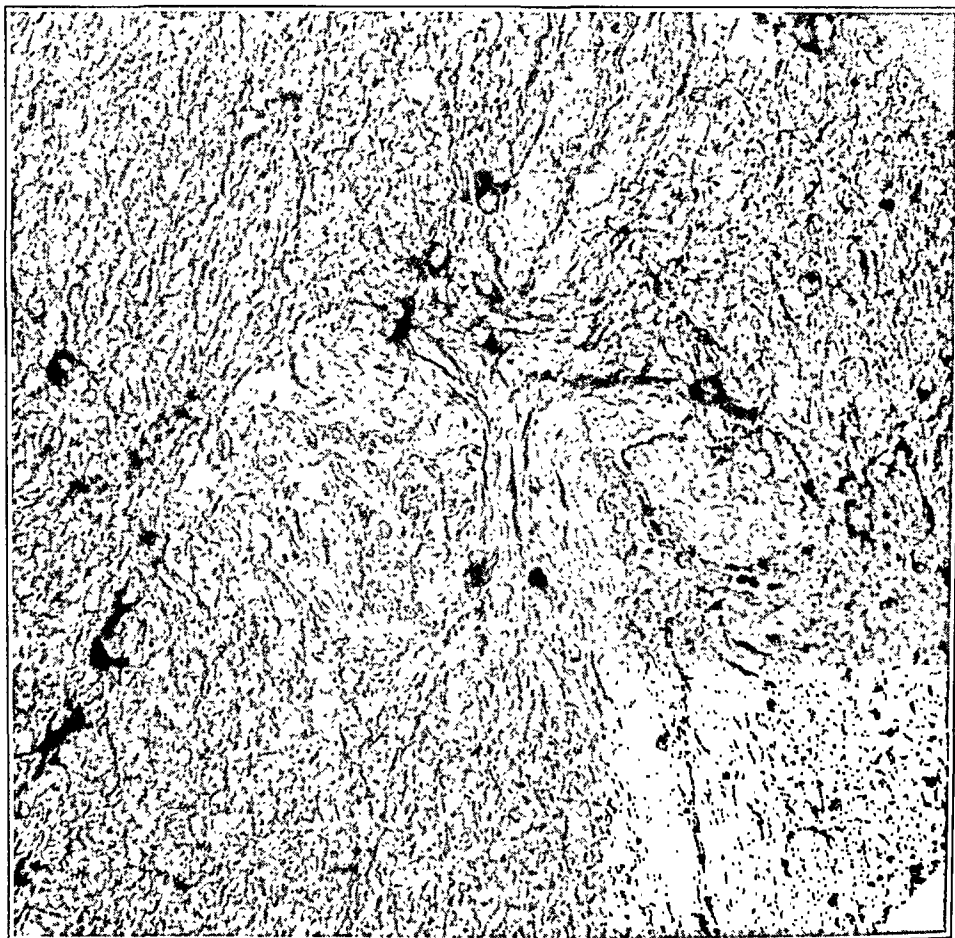


Fig. 28 (case 2).—Section of the same tumor as shown in figure 27. Note the astrocytes and oligodendrocytes.

the tumor. It was fairly normal in appearance, though somewhat thinned in places. Glial cells or fibers invaded it except in a few limited areas.

Small blood vessels appeared throughout the part of the tumor which invaded the nerve sheath, though they were not numerous.

A few cytoid bodies were found in the gliomatous mass invading the sheath. They stained a deep pink and were somewhat elongated and club shaped. With phosphotungstic acid, however, they stained a deep blue-black. Connective tissue elements were conspicuously absent throughout the tumor, except that surrounding the blood vessels and that which made up thickened arachnoid and the remnants of the pia.

Diagnosis.—A diagnosis was made of Recklinghausen's disease of the abortive type, with glioma of the optic nerve (type, astrocytoma) invading the sheath.

Comment.—The previous study of the sister of this patient and the microscopic examination of the tumor of the optic nerve which was removed made the diagnosis relatively simple in this case. Retrobulbar removal of the tumor with preservation of the globe was therefore

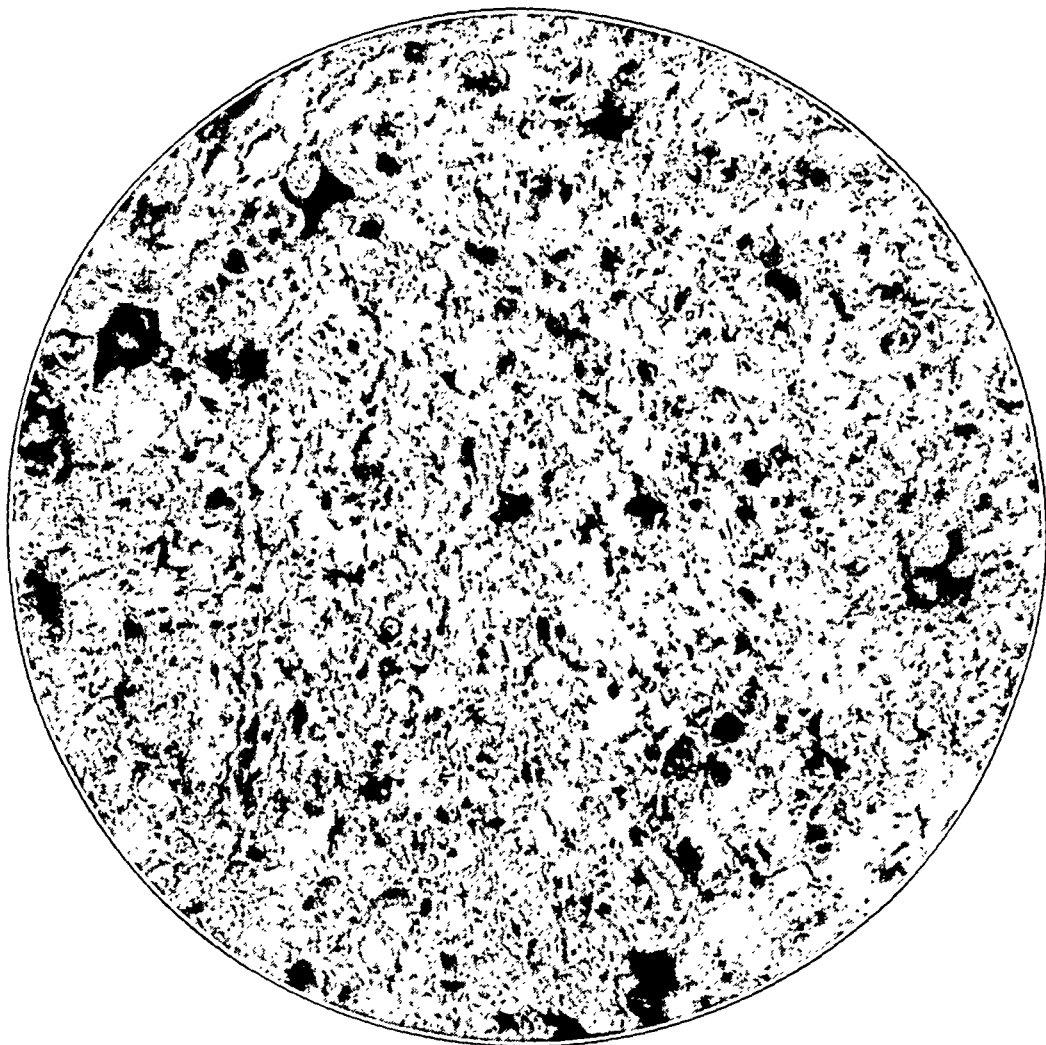


Fig. 29 (case 2).—Multinucleated groups of cells—oligodendrocytes.

decided on. The method of approach was satisfactory in this case as well as in case 3 and is apparently a safer route than that advocated by some others, since the nutrition of the globe was not seriously impaired.³ The excellent postoperative appearance of the eye is note-

3. When this operation was performed I thought that it was an original procedure, since standard texts do not describe it. Subsequent review of the literature, however, revealed that H. Knapp performed a retrobulbar excision of the optic nerve for tumor as early as 1874. Many others have since employed retrobulbar removal of the tumors by various methods other than the well known Krönlein procedure.

worthy, as is the motility of the globe. At a recent examination the general appearance of the eye and the motility remained excellent, although five years have elapsed since the operation.

The postoperative picture of the fundus is of interest, since the retinal vessels contained blood. This no doubt was the result of anastomosis with the ciliary vessels. Pigmentary changes have been

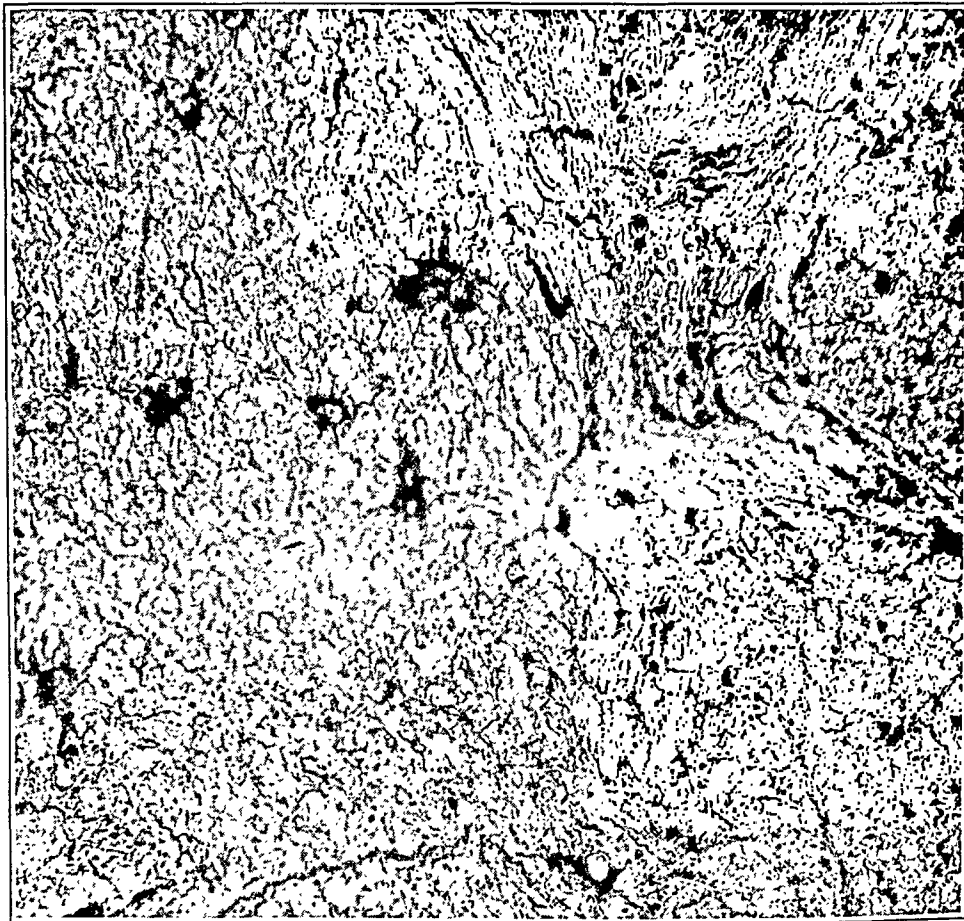


Fig. 30 (case 2).—Same section as in figure 26, showing neoplastic cells.

surprisingly absent. Evidently the ciliary vessels were not seriously injured at operation. This phenomenon has been noted by others, but usually after sectioning of the optic nerve the retinal vessels soon become mere threads and extensive chorioretinal pigmentary degeneration follows, as will be noted in case 3.

The deformity of the fibula and the scoliosis of the spine are of importance, the latter condition having been reported frequently in cases of Recklinghausen's disease (Brooks and Lehmann). The presence of

peripheral signs of Recklinghausen's disease of the abortive type, which was hereditary, is of outstanding significance (figs. 16 and 17).⁴

The tumor in this case was not so far advanced as that in case 1 and therefore afforded an opportunity for study of another stage of glioma of the optic nerve. In later stages of the growth the tumor advances so that it destroys most of the landmarks of the sheath as well as those of the nerve stem itself, so that conclusions regarding the precise

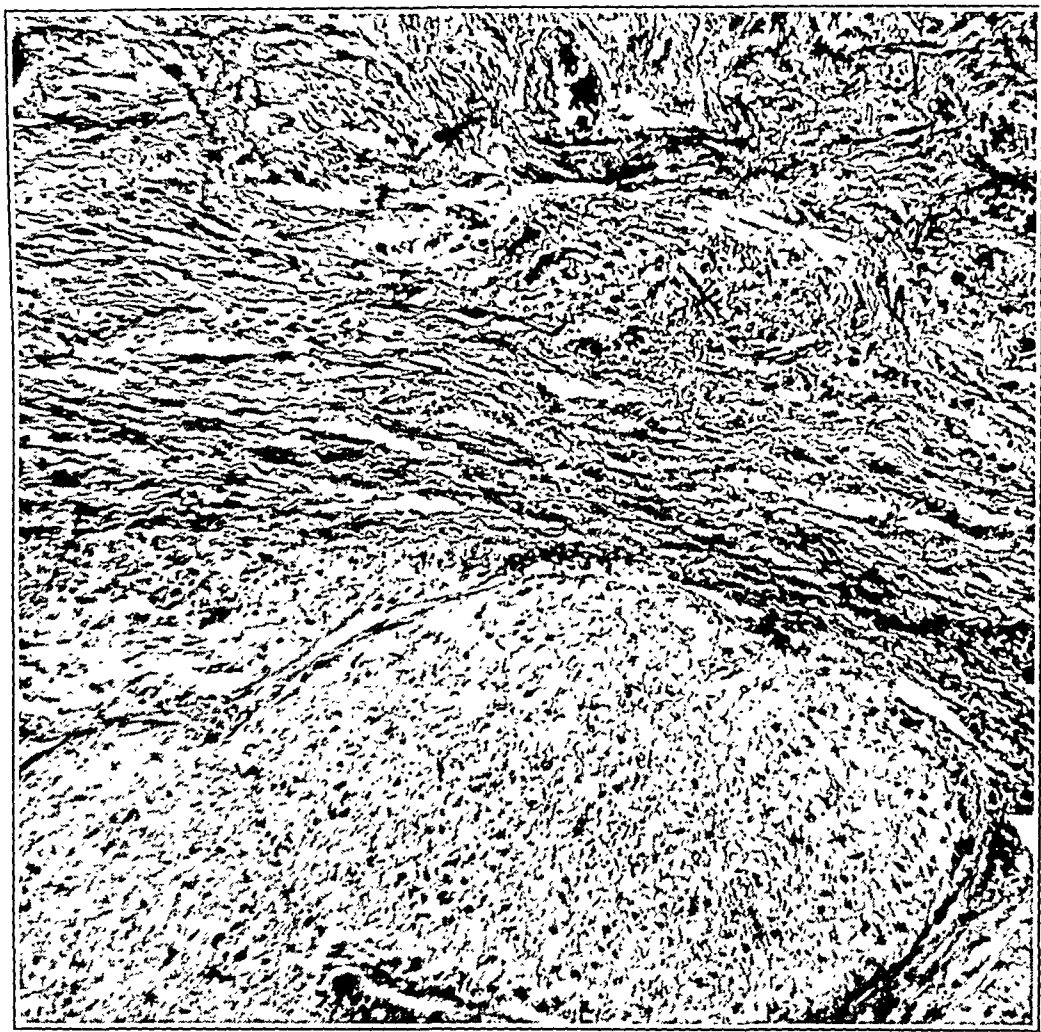


Fig. 31 (case 2).—Higher power magnification ($\times 150$) of the section shown in figure 32. Note that the tumor in the nerve sheath is made up of coarse glial fibers, some straight and some wavy. Also note the almost total destruction of the pial sheath, which has been invaded and broken up by glial fibers. Few cytoid bodies are present. Phosphotungstic acid stain.

4. Slight enlargement of the right optic foramen was difficult to explain at the time the plates were made, since there was no clinical evidence of tumor formation in the right optic nerve. On readmission of the child since the completion of this paper there was definite evidence of an independent tumor developing in the right optic nerve.

nature of its development are rendered difficult—actually impossible in some recorded cases.

The study of this case indicates that the tumor arose within the nerve stem and spread outward to the sheath. This conclusion is more convincing when viewed in the light afforded by studies of the tumor in case 3.

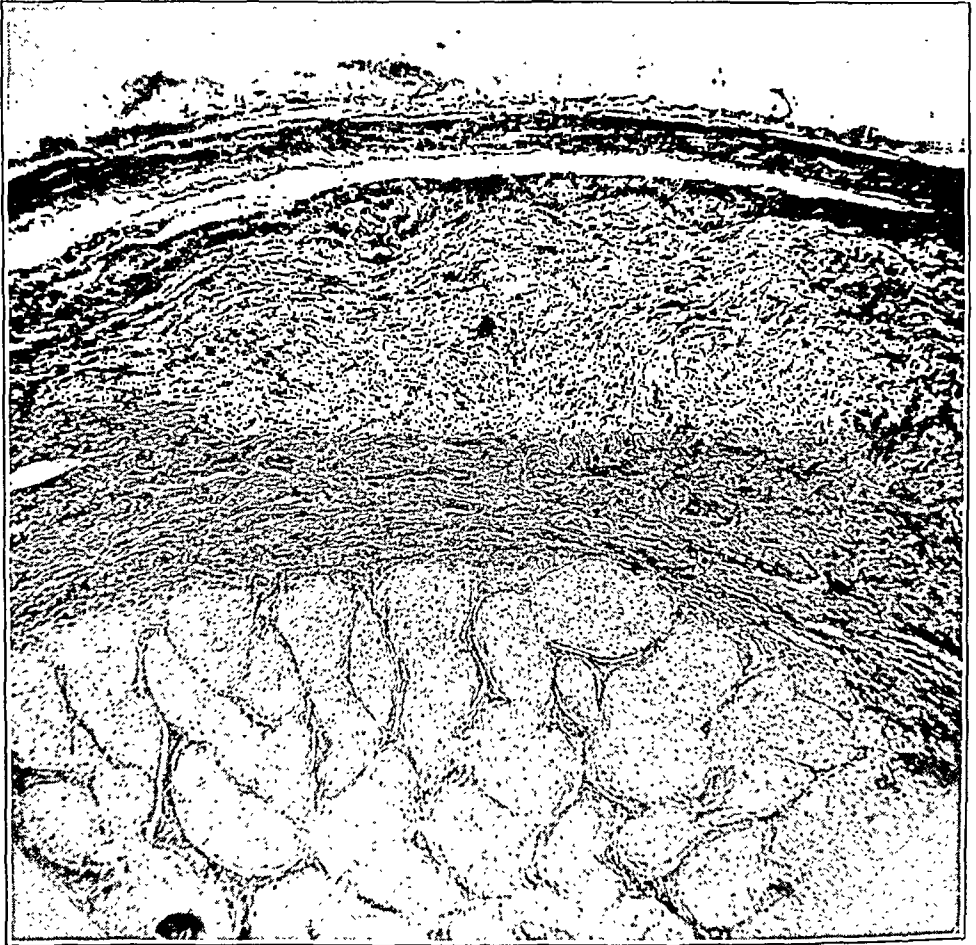


Fig. 32 (case 2).—Transverse section through the nerve stem in the thickest area of the nerve (stage 3; phosphotungstic acid-hematoxylin stains; $\times 80$). Note that the tumor in the nerve sheath is made up of masses of glial cells and fibers which have grown out from the nerve stem; also there is a thin layer of proliferated arachnoid cells beneath the dura (the dura is thin).

CASE 3 (fig. 34).—*Diagnosis of primary tumor of the optic nerve; retrobulbar excision of the anterior half of the optic nerve with preservation of the globe; gliomatosis of the optic nerve (operative specimen) with no involvement of the sheath or enlargement of the nerve; autopsy (four and one-half years later), showing glioma of the optic nerve invading the sheath, gliomatosis of the intracranial portion of the nerve, chiasm, pons and the medulla, a glioma in the temporal*

lobe of the brain, gliomatosis of the fellow nerve and Recklinghausen's phenomena, consisting of cutaneous café au lait-pigmented patches, plexiform neurofibromatosis of the ciliary nerves of the orbit, neurofibromatosis of the choroid, ciliary body and iris.

B. C., a girl aged 3 years, was first admitted to the ophthalmic ward of the Wisconsin General Hospital on Nov. 6, 1933. The child was born in the obstetric ward, there being no complications or unusual findings. She was

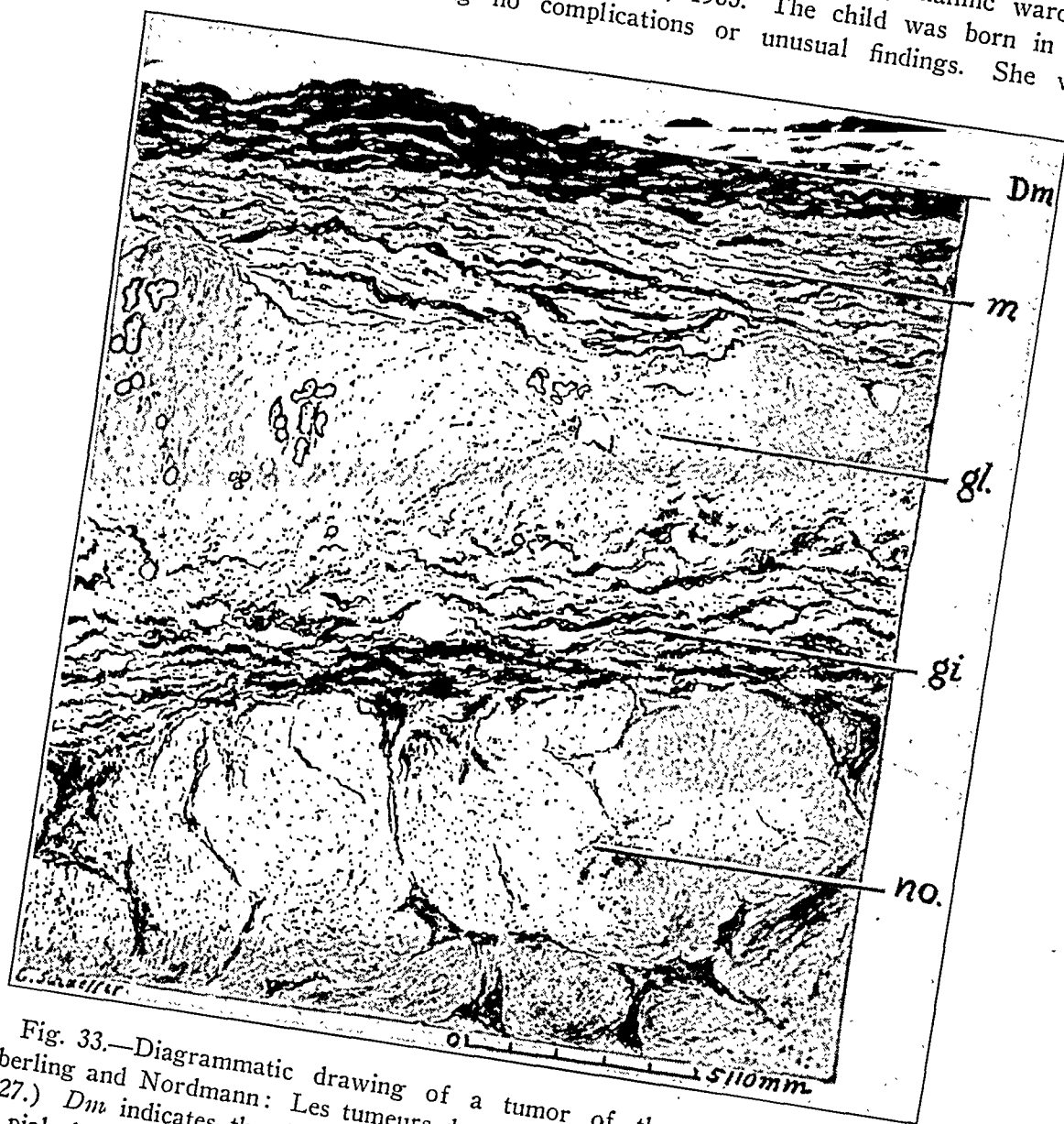


Fig. 33.—Diagrammatic drawing of a tumor of the optic nerve. (From Oberling and Nordmann: Les tumeurs du nerf optique, Ann. d'ocul. **164**:561-660, 1927.) Dm indicates the dura mater; m, meningioblasts; gl, glial proliferation; gi, pial sheath invaded by the glial tumor, and no, glioma within the optic nerve stem. These authors maintain the glial tumors of the sheath arise from the "meningioblasts" from the arachnoid sheath. Compare this figure with figure 32 (stage 3) and with figures 1 and 58, which suggest that the glial tumor of the sheath arises from the nerve stem and not from meningioblasts.

later brought to the pediatric outpatient department for observation because she was pale and had a burn on the leg which healed poorly. Exophthalmos of the right eye was noted at that time, and she was referred to the ophthalmic clinic.

Family History.—The child's mother had typical changes of mild Recklinghausen's disease. There were a number of pigmented patches on the skin and several soft mollusca fibrosa on the back and abdomen and one on the chin. Two of these soft tumors were excised, and microscopic study showed the usual patternless arrangement of the tissues which is found in neurofibromas of this type. The eyes of the mother showed a slight bilateral exophthalmos, which could not be attributed to disease of the thyroid. Examination of the fundi showed slight pallor of the nerve head in each eye. Vision was 20/30. The fields were within normal limits, though there was slight constriction.

General Examination.—Nothing of importance was found on general physical examination aside from a number of pigmented patches in the skin, one on the



Fig. 34 (case 3).—Gliomastosis of the optic nerve. Note the slight exophthalmos of the right eye (4 mm.) and also the slight convergence.

back of the neck measuring 3 by 3 inches (7.6 by 7.6 cm.), two large ones on the thigh measuring $1\frac{1}{2}$ by $1\frac{1}{2}$ inches (3.8 by 3.8 cm.) (fig. 35) and many smaller ones on other parts of the body. They were typical smooth, nonelevated, light coffee-colored spots, which the mother stated were present at birth. These spots were pale and had been completely overlooked or disregarded by three different examiners on three previous occasions.

There was exophthalmos of the right eye, measuring about 4 mm. (fig. 34). Slight ptosis was present, and there was definite limitation of motion in the field of the superior, inferior and external rectus muscles. There was slight convergence. The cornea was clear, and the anterior chamber was of normal depth. The pupil was larger than that of the left eye and was somewhat

irregular and eccentric, but it reacted to light and in accommodation. Examination of the fundus revealed clear media and a pale nerve head with sharp borders, the temporal half showing an unusually large physiologic cupping. Tactile tension was normal.

The left eye was normal.

The results of roentgen examination were reported as essentially normal, but a rereading of the plates later revealed an asymmetry of the bony orbits (fig. 36), the right being definitely larger than the left. The sella turcica itself appeared

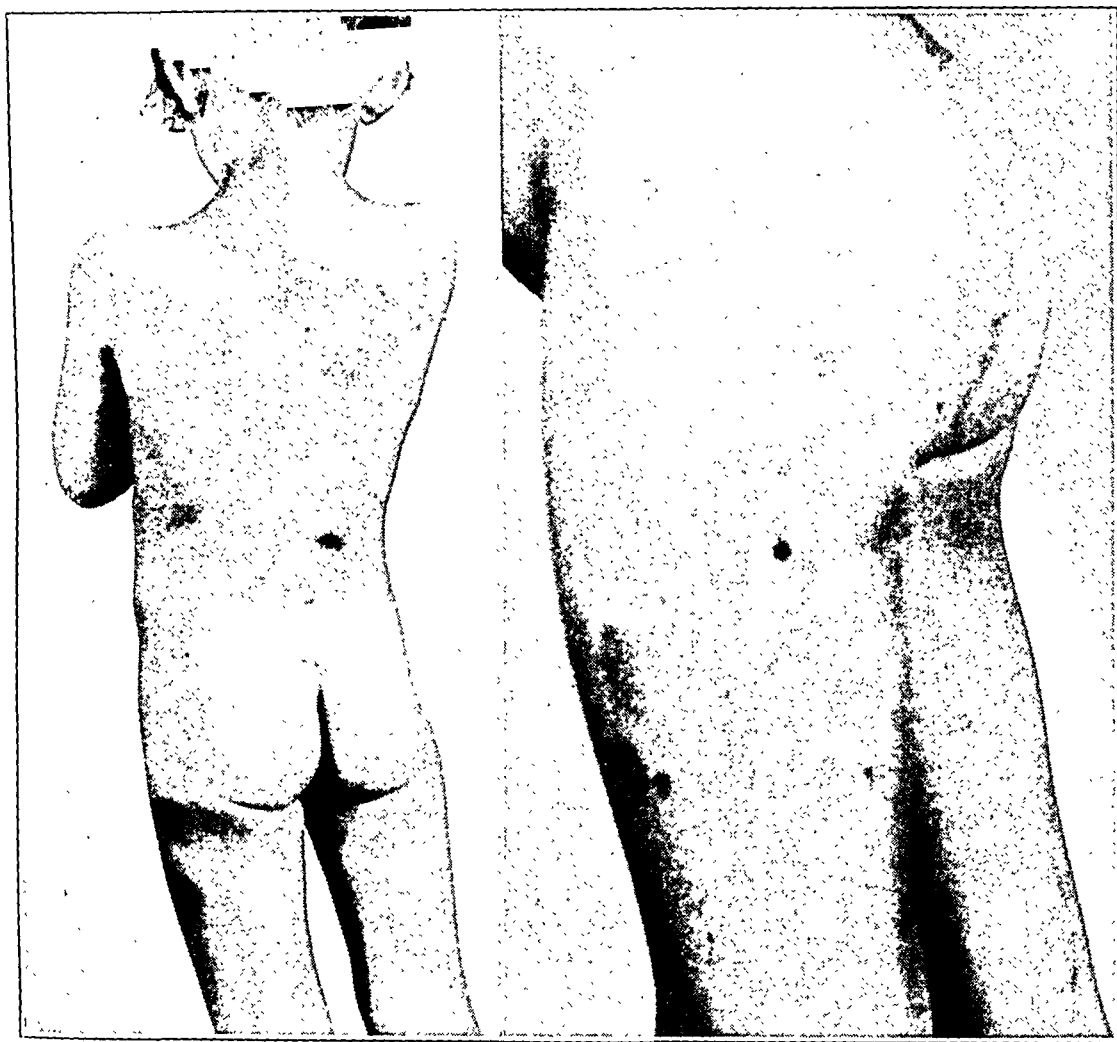


Fig. 35 (case 3).—Café au lait-pigmented patches on the back and leg.

normal, but rereading of the films revealed a small, narrow pear-shaped shadow under the anterior clinoid process.

A tentative diagnosis of tumor of the optic nerve was made, and it was requested that the child be brought in for periodic observation. A second examination three months later (Jan. 10, 1934) revealed what appeared to be a slight increase of the exophthalmos of the right eye, though the exophthalmometer reading was unsatisfactory due to lack of cooperation. The convergence was definitely increased, measuring 5 to 10 degrees, with some limitation of motion but no paralysis of the extraocular muscles. The pupil reacted normally but was somewhat irregular and still eccentric, being drawn toward the temporal side.

The iris showed definite atrophy. It was somewhat thinned and had an ironed-out appearance. It dilated to 7 mm. with homatropine hydrobromide. The media were clear. The disk was seen with a —2.00 sphere, and the temporal pallor had increased and was to be seen in the nasal half of the disk as well as in the temporal portion. The cupping was considered physiologic, though it was unusually large and deep, and so was regarded with suspicion. The vascular system was normal. The vision was estimated with difficulty, owing to the age of the patient, but that of the right eye appeared to be definitely reduced.

Roentgen examination showed the same asymmetry in the size of the orbits as previously noted as well as the anterior necklike shadow under the anterior

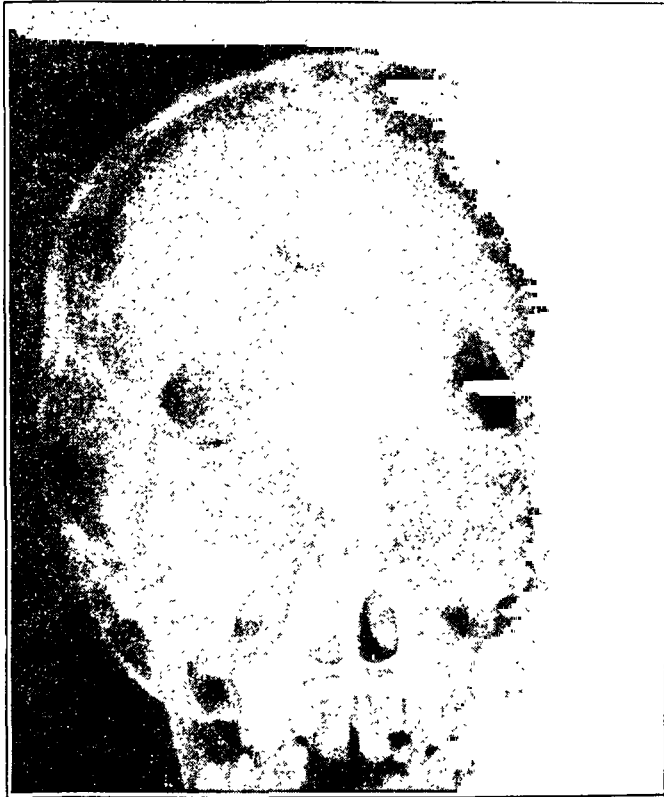


Fig. 36 (case 3).—Roentgenogram of the skull. Notice the enlargement of the right bony orbit, which was due to plexiform neurofibromatosis of the nerves of the orbit.

clinoid process (fig. 37). Stereoscopic plates revealed no abnormality of the body of the sella turcica.

The left eye was normal, though the disk appeared slightly pale.

A diagnosis of tumor of the right optic nerve was made, and operation was advised.

Operation (Jan. 31, 1934).—Anesthesia was produced by the endotracheal administration of nitrogen monoxide and ether. Exploration of the right orbit was done as outlined in case 2, through an incision in the skin along the lower bony rim, the orbit being entered directly through the septum orbitale (fig. 38). The nerve was exposed with greater difficulty than in case 2. At the time of operation this

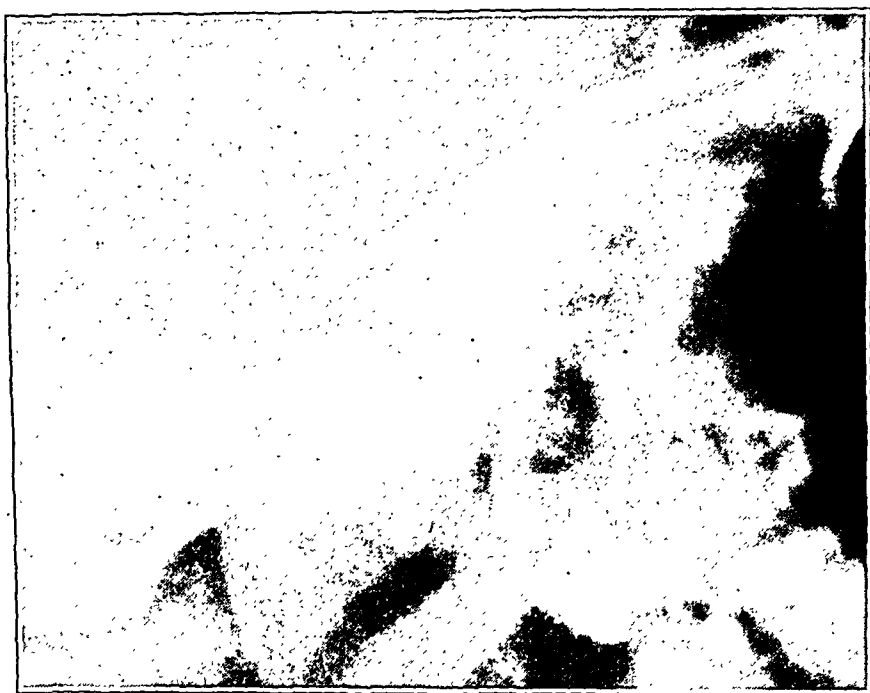


Fig. 37 (case 3).—Sella turcica (actual size). Note the pear-shaped shadow which projects forward from the upper anterior aspect of the body of the sella under the anterior clinoid process.



Fig. 38 (case 3).—Appearance of the patient one month after operation (retrobulbar excision of one half of the optic nerve). Note the line of incision in the lower lid; this disappeared after complete healing (see figure 43).

was attributed to the smaller size of the bony orbit in a child of 3 years of age. The nerve was tied off with catgut where it joined the globe and also near the middle of the orbit, and a section about 11 mm. in length was excised. The wound was closed as described in the previous case, and the lids were sutured together with a mattress suture, since I anticipated some trophic change in the cornea.

There was moderate postoperative reaction and swelling, though not nearly so marked as in case 2, and healing progressed so that the dressing was removed in three weeks. The suture was removed from the lids on the tenth day after operation. The cornea was found to be clear and the pupil fairly well dilated from atropine, which had been used daily. The action of the lids was good, and the extraocular movements, which had been absent, began to return feebly. The child was discharged four weeks after operation and was observed from time to time in the outpatient department.

Microscopic Examination (section of the optic nerve removed at operation).—Grossly the nerve appeared of about normal size throughout the portion excised, measuring about 3 mm. in diameter (fig. 39). Sections revealed no thickening

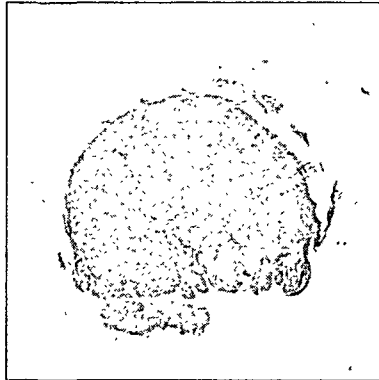


Fig. 39 (case 3).—Transverse section through the optic nerve ($\times 11$). Note that the sheath is not involved. The dural sheath was injured on removal of the nerve.

of the dura or arachnoid, nor was there any infiltration in the intervaginal spaces in any part of the nerve. The pia was intact, but both the arachnoid and the dura were considerably thinner than in the normal preparations, probably due to trauma in isolating the nerve from surrounding structures. Transverse sections of the nerve (fig. 40) appeared strikingly normal to casual inspection. The general architecture of the nerve was preserved, though there was some enlargement of the funiculi. Careful study revealed a vacuolation of the tissue within the funiculi and condensation of glial fibers which bordered the fibrous septums (fig. 41). Staining with phosphotungstic acid and hematoxylin showed a marked increase in glial fibers, especially adjacent to the septums. The glial cells throughout the funiculi were increased both in size and in number. Vacuoles which appeared throughout the funiculi were found to be due in part to vacuolation of the glial cells and in part to actual holes. The latter appeared to be attributable to shrinkage or to complete atrophy and disappearance of the nerve fibers. A few of the myelin sheaths were present in some areas, but they had completely disappeared throughout most of the sections examined. The vacuolation of the tissue

within the funiculi was similar to that observed in the sections in cases 1 and 2, though not so extensive, while formation of glial fibers was not so advanced.

Subsequent Clinical Data (July 23, 1934).—Six months after operation, according to notes made at the time, there was still slight exophthalmos with some widening of the palpebral fissure. The incision in the skin showed a faint linear scar. There was some fulness above and below the orbital rim. Ocular motility was free in all fields, with a tendency to convergence at times—about 5 degrees, as seen before operation. The globe was somewhat larger than that on the left side. The cornea remained clear; it appeared slightly larger than that of the left eye. The anterior chamber was of fair depth; the aqueous was clear; the

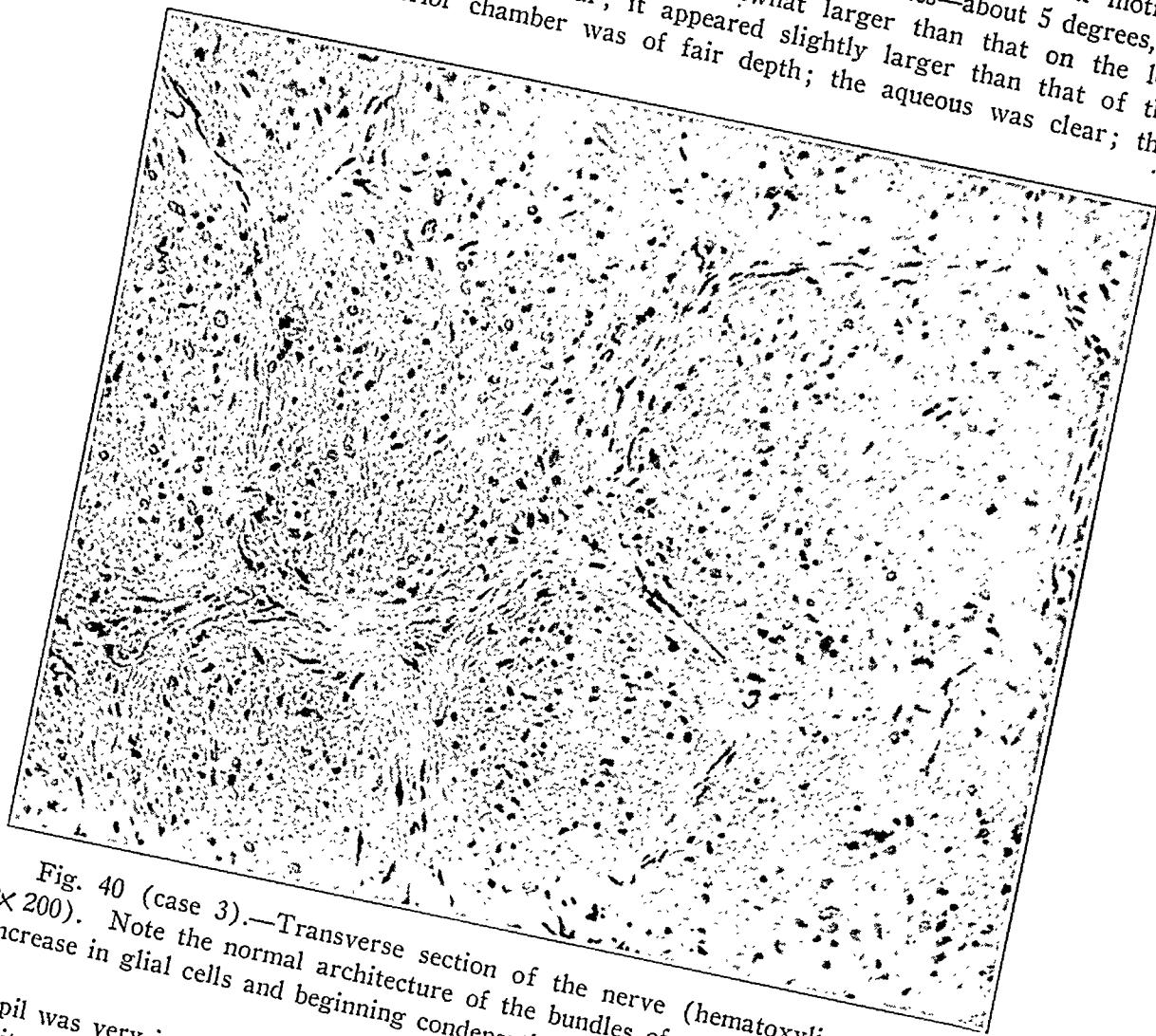


Fig. 40 (case 3).—Transverse section of the nerve (hematoxylin and eosin; $\times 200$). Note the normal architecture of the bundles of nerve fibers, the marked increase in glial cells and beginning condensation of fibers bordering septums.

pupil was very irregular and displaced temporally upward, measuring about 5 mm. in its longest axis, and the iris appeared atrophic. There appeared to be a slight consensual reaction to light; the media were clear; the fundus showed extensive degeneration of retinal pigment, and large masses appeared throughout the retina, one mass lying directly over the disk and obscuring its outline, though its pallor could be seen. The vessels were narrow and apparently bloodless.

A note made one year later (July 5, 1935) stated that there was little change in the eye since the previous examination. There appeared to be slight swelling of the lids of the right eye. Exophthalmos was slight and unchanged from that last recorded. The cornea was clear; the extraocular movements were

normal, and the anterior chamber was a little shallower than normal. The pupil was very irregular and displaced temporally and upward; it measured 5 mm. at its longest axis and was pear shaped (figs. 42 and 43). The iris was atrophic; the fundus appeared as previously described.

Examination on March 16, 1936, showed little if any change from that of July 23, 1934. Lateral stereoscopic roentgenograms of the sella turcica were again made and revealed a necklike, pear-shaped shadow extending from the

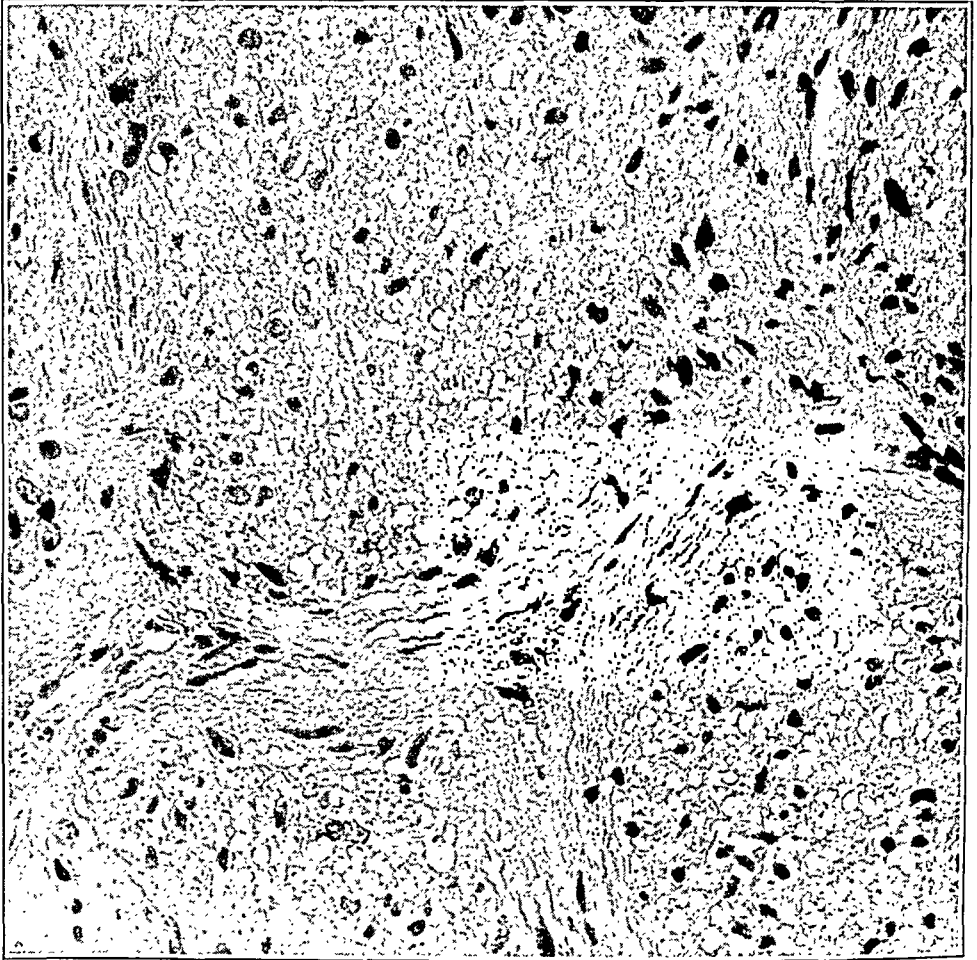


Fig. 41.—High power magnification of the section in figure 40. Note the vacuolation of glial cells and the holes in the glial framework of the nerve. The nerve fibers had largely atrophied.

upper edge of the body of the sella turcica under the anterior clinoid process. The optic canals were reported about equal in size and shape and within normal limits in diameter. The cranial bones were thin.

On Sept. 16, 1936, two and one-half years after operation, examination revealed slight exophthalmos and fulness of the lids of the right eye. There was a faint scar in the lower lid at the line of suture. The cornea was clear; the anterior chamber was deep; the pupil was larger than the left and still eccentric; the media were clear; the fundus showed extensive pigmentary degeneration of the

choroid and retina, and no retinal vessels were visible. The optic nerve showed advanced atrophy and was partially covered by a mass of pigment. The left eye appeared normal aside from a definite pallor of the nerve head, which had increased



Fig. 42 (case 3).—Photograph was taken four and one-half years after operation at the age of 7. The right cornea is slightly larger than the left. Note the pear-shaped coloboma of the pupil due to contraction from neurofibromatosis of the iris. There is paresis of the left facial nerve. Note the excellent position of the globe and the faint line of scar.

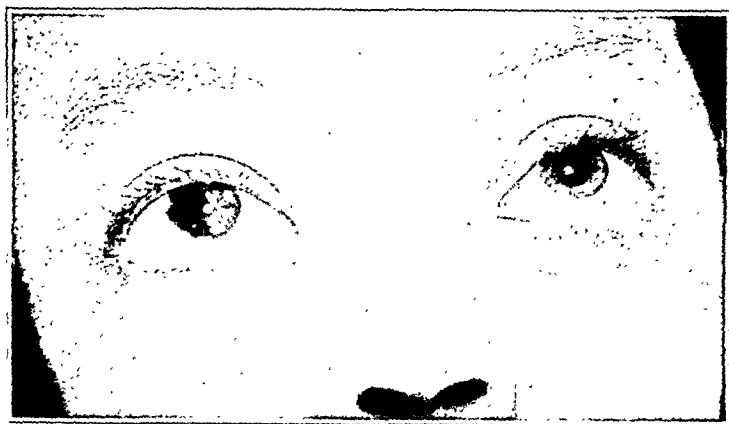


Fig. 43.—Closer view of the patient's eyes, showing distortion of the pupil of the right eye resembling a pear-shaped coloboma. Note the faint line of scar in the skin of the right lower lid—the site of orbital exploration.

since the previous examination. Vision in the left eye was 20/30 — 2 with a + 1.00 sph. \ominus + 1.50 cyl., axis 90. The school nurse reported that the child was having difficulty with close work in school.

The child was readmitted to the hospital for study six months later (Feb. 25, 1937). At that time slight exophthalmos of the right eye was noted (2 mm. greater than the fellow eye), with definite fulness of the upper and lower lids. The line of incision made at operation was barely visible. Nothing could be palpated in the orbit. The eyes were straight, and ocular movements were free in all fields. The cornea of the right eye remained clear, and the anterior chamber was of fair depth. The pupil was more drawn to the temporal side and measured 4 by 5 mm. A consensual reaction to light seemed to be present, though slight. The pupil dilated well with hematropine hydrobromide. The iris appeared atrophied. Examination of the fundus revealed a few coarse floating bodies in the vitreous. The lens was clear. The fundus picture had changed little. The color reflex was pale, but the entire retina was covered with patches of pigment. Some white patches were seen which appeared like masses of glial tissue or scar tissue. The retinal vessels were absent, only a few faint, thin remnants in the form of fibrotic threads being visible. The disk was indistinctly seen and appeared white. It was obscured by masses of retinal pigment.

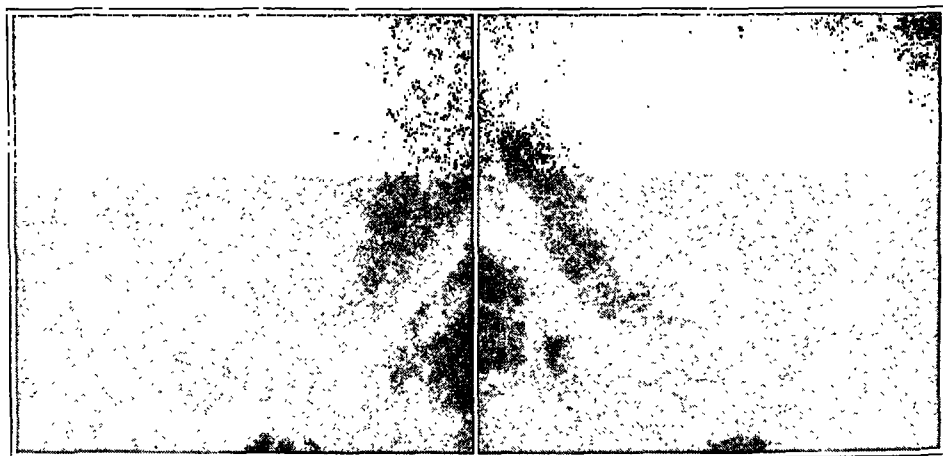


Fig. 44 (case 3).—Roentgenograms of the optic foramina (actual size). Both foramina are slightly enlarged, that of the right side being definitely the larger. (The photographs were taken one and one-half years after the first observation.)

The left eye appeared normal aside from definite atrophy of the optic nerve. The disk was oval and flat, and the atrophy was considered grade 2. The vessels appeared normal. The vision was 20/30 — 2.

Roentgen examination showed that the asymmetry in the size of the orbits, which was present in the original films, had remained unchanged. Roentgen studies of the optic foramina showed definite changes, the right foramen being larger than the left. Measurements made on the films (fig. 44) showed the right optic foramen to have a transverse diameter of 7.5 mm. and a vertical diameter of 6.8 mm.; the transverse diameter of the left optic foramen was 6.8 mm. and the vertical diameter 5.6 mm.

There was some stippling of the bones of the vault.

The pigmented patches in the skin showed no change. The child appeared to be in good health.

Brief observation in the clinic three months later (Jan. 3, 1938) showed the visual acuity in the left eye to be 20/100. Owing to my absence at this

time, no special studies were made. The child was next seen on March 10, 1938, which was about one year after I had last seen her. She was admitted to the wards for further study. The patient, now aged 7, had developed physically as a normal child, though her mentality was definitely below that of children of her age. Her mother reported that she had vomited at intervals for several weeks. The vomiting had been sudden and projectile in type. She had not been ill otherwise and did not complain of headaches. Neurologic examination revealed paresis of the left facial nerve, central in type, with exaggerated knee jerks and a suggestive Babinski sign.

Examination of the right eye showed slight exophthalmos, which measured 3 mm. greater than that of the left eye. There was also slight ptosis. The extraocular movements were normal. The cornea was clear except for a faint opacity in the outer layers of the stroma extending across the lower third. The anterior chamber was slightly deeper than in the left eye. The pupil was pear shaped and drawn to the temporal side (fig. 43); it was inactive to light. The iris was abnormal, showing definite atrophy. Its markings were obliterated and its color faded and blotchy. The lens was clear. There were a few floating shred-like opacities in the vitreous. The fundus showed extensive chorioretinal pigmentary degeneration, as previously described. Tension appeared normal in each eye to touch. Vision in the right eye was nil.

The left eye appeared normal aside from fairly advanced atrophy of the nerve head. The outlines of the disk were sharp and distinct. Vision was 20/100. The field appeared somewhat contracted, though accurate measurement was difficult due to poor cooperation.

Roentgenograms of the skull revealed definite changes when compared with films made the previous year. Lateral views showed a marked increase in the size of the necklike shadow which extended anteriorly from the upper rim of the body of the sella turcica under the anterior clinoid process (fig. 45). When the skull was viewed in perfect lateral position, there was a large, bulbous, slightly pear-shaped shadow; when the head was slightly tilted, a double shadow was seen (fig. 45). The shape of the body of the sella turcica was unchanged from that revealed at the previous examination, though it and the skull as a whole were larger, due to natural growth. The skull showed digital markings throughout the vault, suggestive of intracranial pressure (fig. 45). The right orbit remained unchanged. The right optic foramen had increased slightly in size.

A diagnosis of intracranial tumor was made. It was considered likely that the tumor in the unremoved portion of the optic nerve had extended back into the chiasm. The patient was therefore referred to the department of surgery. Further studies by Dr. E. R. Schmidt, which included a ventricular puncture (April 7, 1938), confirmed this diagnosis. A ventriculogram (read by Dr. W. J. Eleckwenn) revealed the left ventricle to be larger than the right. No third ventricle was visualized. In the lateral view the right ventricle showed a defect, the temporal horn being cut off near the ampulla. The lateral body of the ventricle was pushed upward, giving the impression of a lobulated tumor in the right temporal lobe. There was a slight shift of the right ventricle to the left. The reading suggested a subcortical tumor.

Operation (April 30, 1938).—Dr. E. R. Schmidt performed a right frontoparietal craniotomy. The right optic nerve was exposed and found to be considerably enlarged. A tumor was encountered in the anterior and medial portion of the right temporal lobe. The chiasm appeared slightly enlarged, but no definite tumor was found. The tumor being inoperable, the right optic nerve extending into the chiasm was removed, as was a small section of the tumor. After this

considerable bleeding developed, which was promptly controlled by electrocoagulation. The patient was given a transfusion on the operating table. The bone flap was replaced and the wound closed. The patient was returned to the ward and died ten hours later.

General Autopsy (Dr. J. L. Parks).—A complete postmortem examination was made, and the right eye and orbital contents were removed. The general observations included postmortem ulceration and perforation of the stomach and left side of the diaphragm, digestive necrosis of the peritoneum, omentum and left pleura and pancreatic necrosis. The pituitary body showed hemorrhage and necrosis in the anterior lobe.

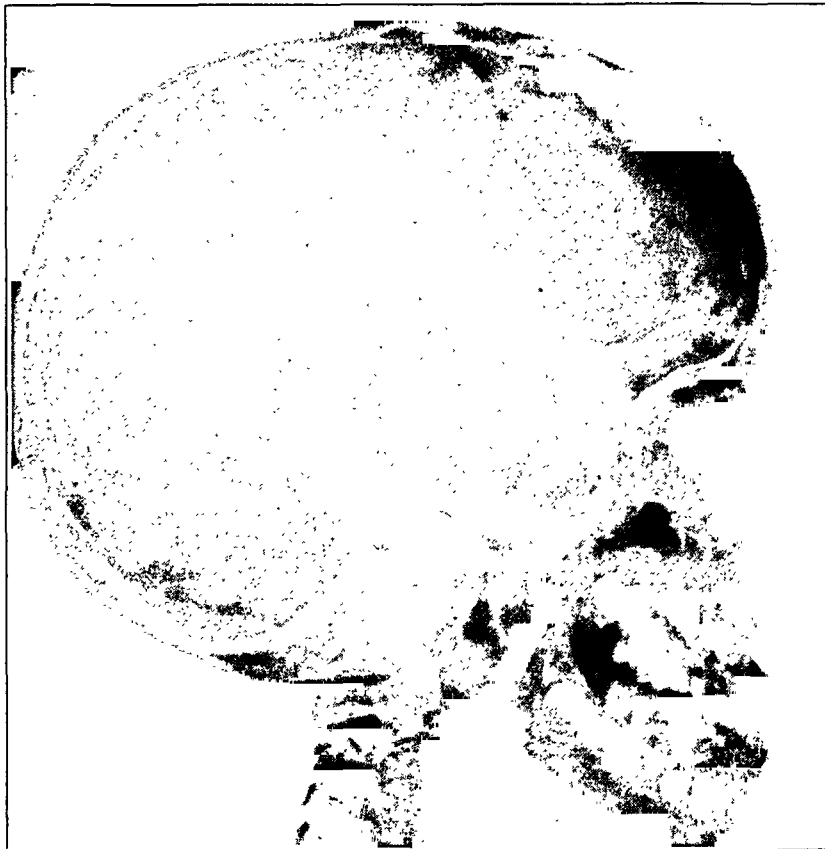


Fig. 45 (case 3).—Roentgenogram of the skull taken four years after the first observation. Note the increased depth of the sella turcica and marked increase in the size of the pear-shaped shadow. There are digital impressions in the vault, due to increased intracranial pressure.

Macroscopic Study of the Brain (Dr. J. C. McCarter).—According to the report made, there was a gliomatus tumor measuring about 4 by 4 by 3 cm. in the anterior and medial portion of the right temporal lobe. The tumor was firm and gray and showed some irregular cystic softening. It had fairly clear borders but was not encapsulated (fig. 46).

The tumor had destroyed and presented to the surface in the region occupied by the hippocampal gyrus. It compressed the inferior convolutions of the temporal

lobe and compressed and pushed laterally the inferior horn of the right lateral ventricle. It partially destroyed the inferior portion of the right lenticular nucleus and approached but did not invade the posterior limb of the right internal capsule.

The hypothalamic region was covered with clotted blood, and the relations of the optic nerves and chiasm could not be clearly made out. The chiasm did not appear to be especially enlarged.

The posterior portion of the tumor caused pressure across the fissure between the temporal lobe and cerebral peduncles, and the optic tract on the right could

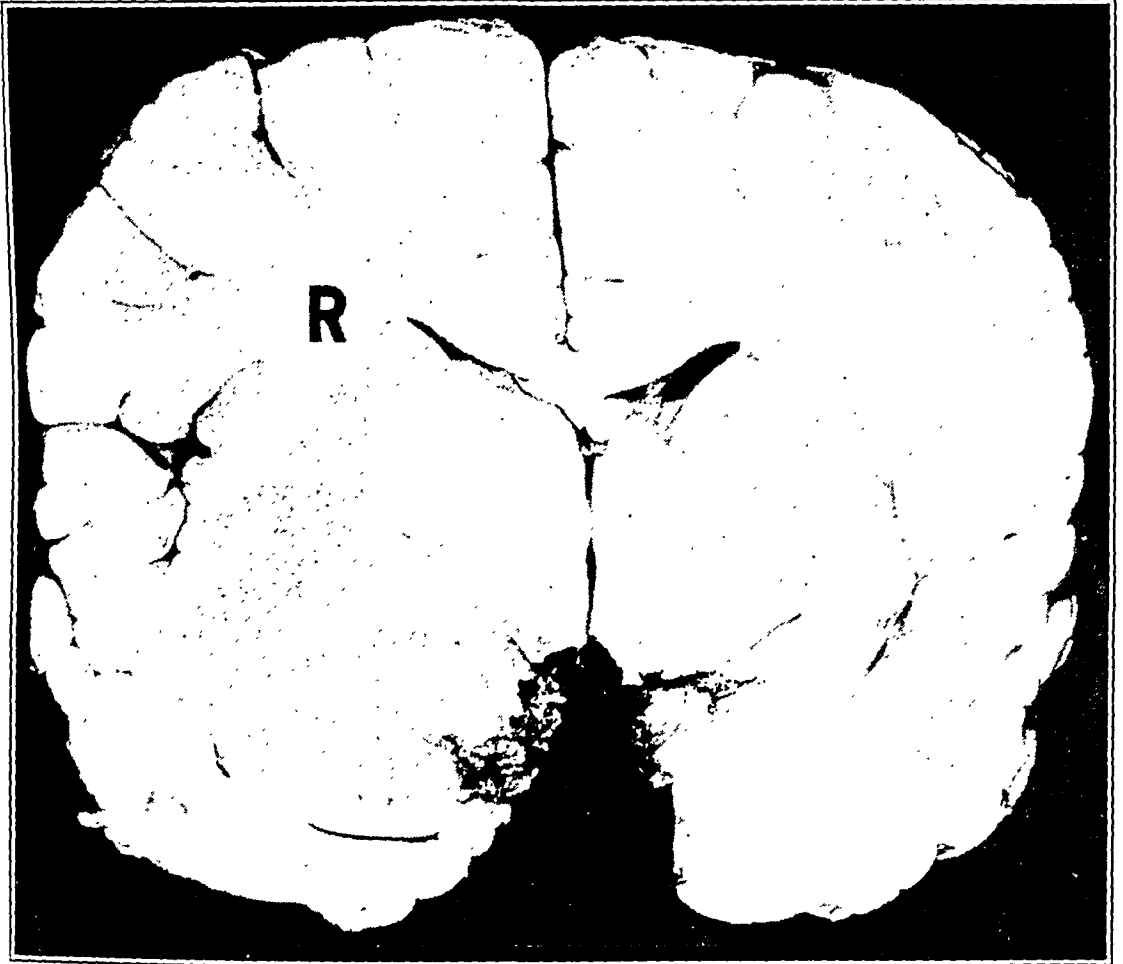


Fig. 46 (case 3).—Section of the brain. Note the tumor (glioma) in the left temporal lobe.

not be identified owing to atrophy from this pressure. The optic tract on the left was identified, but appeared atrophic with minute cystic changes.

The corpus striatum, the thalamus, the pons and the medulla oblongata were definitely enlarged and extremely firm. These regions were pale, and the normal contrast between gray and white matter was much reduced.

There was evidence of recent trauma (evidently due to operation) about the tip of the right temporal lobe, about the optic chiasm and at the base of both frontal lobes in the olfactory trigons.

Microscopic Study of Brain Tissue (Dr. J. C. McCarter).—A cross section area of the medulla (fig. 47) was about twice the normal size for an adult. The nerve cells were few in number and sparse in distribution, and many remaining ones showed cloudy swelling and chromatolysis. No demyelination was demonstrated. There was a striking increase in the number of astrocytes with the formation of many fibrils, which were isomorphic in distribution.

The changes in the pons were similar to those in the medulla. Several giant astrocytes were seen.

The cerebellum showed some increase in astrocytes in the white matter.

The changes in the thalamus were similar to but not as great in degree as those in the medulla.

The chiasm showed changes similar to those noted in the medulla, though the chiasm was but slightly enlarged. Small atrophic areas replaced by vacuolated tissue were seen. The nerve cells in the adjoining hypothalamus showed swelling and chromatolysis.

The tumor was an astrocytoma, showing evidences of increasing malignancy and less differentiation (fig. 48).

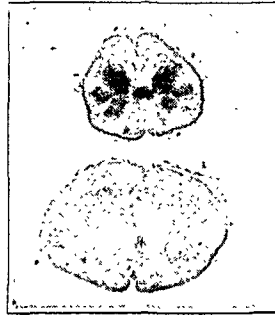


Fig. 47 (case 3).—Upper section, medulla from a normal adult, actual size. Lower section, enlarged medulla with diffuse gliosis; also actual size.

The tumor showed marked cystic degeneration in which large spaces were filled with a clear, structureless material. Much of the tumor was so necrotic that it stained poorly by any method. The cells appeared of mixed types, some having the appearance of unipolar and bipolar spongioblasts and others resembling swollen protoplasmic astrocytes.

Study of the Eye and Orbit, Region of the Hypothalamus, Chiasm and Optic Nerve.—The eye, together with the surrounding soft parts of the orbit, was removed at autopsy and placed in a 10 per cent dilution of formaldehyde. The specimen was cut transversely in three parts after the globe was severed. One section was mordanted for Weigert staining, one was refixed in Zenker's solution and the third was left in formaldehyde.

No satisfactory macroscopic study could be made, since I did not wish to disturb the relation of the tissues about the nerve. The cut end of the nerve at the apex of the orbit appeared definitely enlarged to about twice its normal size, with some thickening of the sheath. One part of the nerve (approximately one-third on cross section) on gross inspection appeared darker in color than the other two-thirds. The chiasm and region of the hypothalamus were slightly

enlarged but showed no definite tumor formation on gross inspection. The right optic nerve and a small section of the chiasm were missing from the postmortem specimen, since they were removed at operation. A short section of the left optic nerve was seen as it entered the chiasm. The intracranial portion of the nerve removed at operation was fixed in formaldehyde-ammonium bromide as well as in Zenker's solution. It appeared to be about twice the normal size but showed no gross evidence of tumor about the sheath.



Fig. 48 (case 3).—Section from the tumor of the brain.

Microscopic Study of the Optic Nerve: Part of the operative specimen of the nerve was sectioned after freezing; the remainder was embedded in paraffin. The postmortem specimen, including the eye and orbital contents as well as the chiasm, was serially sectioned. Various methods of staining were used, including hematoxylin and eosin, Van Gieson's stain, Mallory's phosphotungstic acid-hematoxylin stain, Mallory's trichrome stain, Weil's iron hematoxylin, Weigert's myelin sheath stain, the Pal-Weigert stain, Cajal's gold chloride stain, the Hortega and Weil-Davenport ammoniacal silver methods for astrocytes and oligodendrocytes, and others.

The intracranial section of the optic nerve, removed at intracranial exploration, included a small part of the chiasm where the nerve entered it. The nerve was oval and about twice its normal size. It showed marked proliferation of the glial cells, with some fiber formation. These fibers were grouped about the connective tissue septums and the pia. The pia was intact and showed only slight invasion by glial fibers in a restricted area. The arachnoid, where present, showed no abnormal proliferation or thickening, such as was noted in the intraorbital part of the nerve. The histologic picture bore a striking resemblance to the sections of the nerve removed at the first operation.

The sections of the chiasm, optic tracts and the stub of the left nerve were serially sectioned, but for some reason staining was not satisfactory. Marked glial proliferation of the right nerve, which adjoined the chiasm, was found, the sections staining well with various methods, with the exception of the silver methods. Here, though the specimens were cut from frozen sections, the technic was not entirely satisfactory. There appeared to be moderate glial hyperplasia throughout this region but no evidence of tumor formation.

The intraorbital part of the nerve (autopsy specimen) showed considerable variation, depending on the level at which the nerve was sectioned. Serial sections revealed that the main body of the tumor occupied a position a little posterior to the point where the nerve had been cut at the original operation four years previously. The nerve stem was slightly oval and measured 4 by 7 mm. (slide section). Surrounding this, the sheath was thickened and increased the diameter over all to 6 by 9 mm. The size gradually diminished toward the apex of the orbit, where the nerve stem itself was about half again the size of the normal nerve, with a corresponding thinning of the part of the tumor which invaded the sheath.

Anterior to the cut end of the nerve was a tangled mass of connective tissue, which filled the space formerly occupied by the nerve. This extended up to the globe. It contained numerous ciliary nerves, for the most part cut transversely. These were much enlarged and showed varying stages of proliferation of tumor cells. The appearance was fairly typical of that generally described as plexiform neurofibromatosis (fig. 49). As the sections approached the globe, the tissue was made up largely of a tangled mass of connective tissue and strands of collagen. There was no remnant of the original optic nerve, since it had been excised.

Where the sections approached the cerebral end of the nerve, which was not excised at the original operation, traces of the sheath first appeared. The dural sheath was first identified, and sections gradually disclosed its normal contour. It appeared fairly normal, though thickened. Lying within the lumen of this dural envelop there was a mass of proliferated arachnoid cells. As the sections extended farther back toward the apex of the orbit, traces of the nerve stem began to appear. This consisted of dense masses of glial fibers; it stained deep blue and was embedded in a meshwork of pink-staining tissue (phosphotungstic acid and hematoxylin), which apparently arose from the pial lamellae. These bands of fibers criss-crossed in no definite pattern and had the appearance of collagen, no definite nuclei or cells appearing in them. Surrounding this there was a thick layer of proliferated arachnoid cells which was sharply limited in a bandlike sheath. Isolated cells were typical flattened ones with short, oval nuclei, though some showed marked enlargement of the nuclei. This area of proliferated arachnoid cells stained light pink in sharp contrast to the dense blue of the glial elements within the nerve stem and pia, and no glial fibers penetrated it. As the sections were followed back, the architecture of the nerve and its enveloping sheath

assumed a more orderly arrangement, but the nerve stem and surrounding tumor gradually increased in size (figs. 50, 51 and 52).

Microscopic study of sections from the thickest area of the tumor revealed the following picture: The nerve stem showed the usual arrangement of the funiculi separated by connective tissue septums (fig. 53). The funiculi, however, were markedly enlarged and their shape distorted. The septums were present, while the intraseptal spaces were compressed in places and distended in others. The most striking feature of the section was the enormous overgrowth of glial cells and fibers. The fibers occupied and filled the entire area of the funiculi. They were most

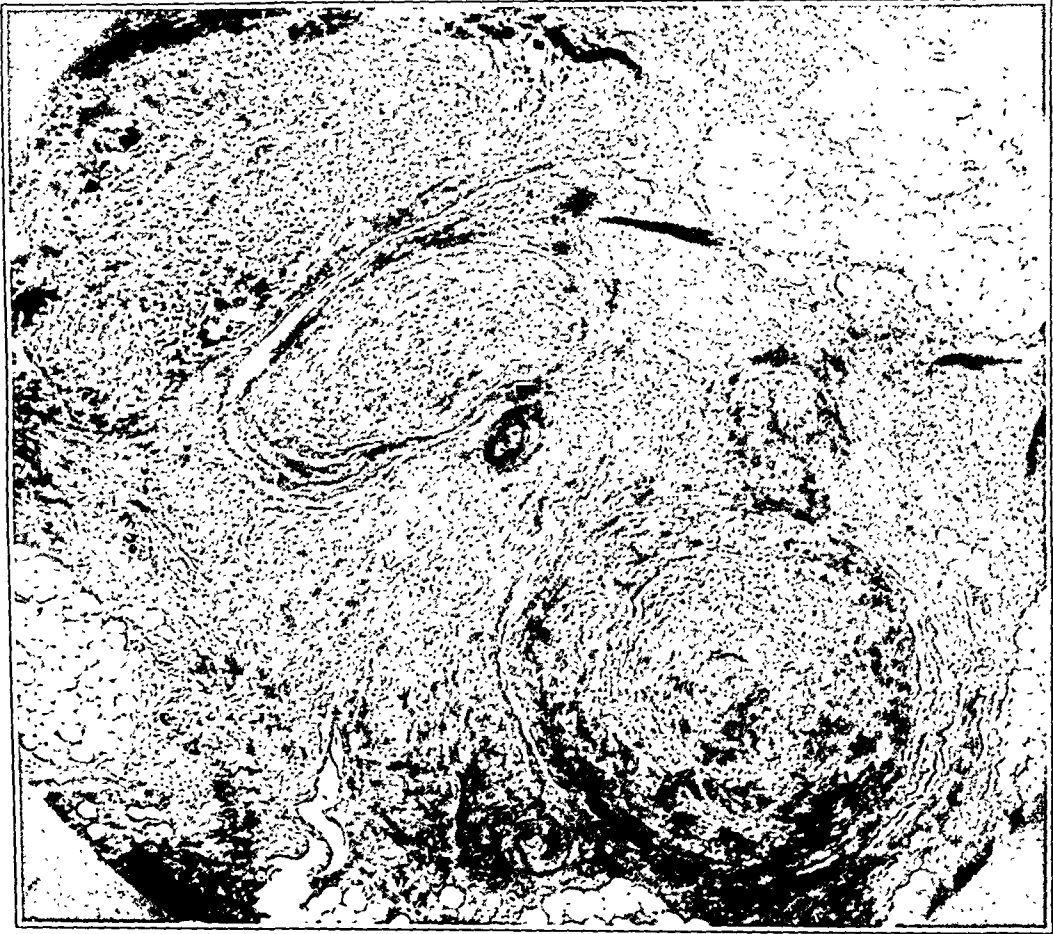


Fig. 49 (case 3).—Plexiform degeneration of the nerves of the orbit. Note the three large degenerated nerves.

dense about the borders of the funiculi, where they abutted the connective tissue septums (fig. 54), but they were not so sharply limited to this area as those in the original section of the nerve or those in the sections described in cases 1 and 2. The glial fibers invaded the intraseptal spaces in many places, though they did not destroy them. No trace of nerve fibers could be found by either Weigert's or Weil's stain.

The nuclei of the cells showed the same variations as those described in the original operative specimen and in sections from the specimens in cases 1 and 2. These were large oval and round forms, with some of irregular contour, especially

imperfectly oval (figs. 55 and 56). Many of these oval nuclei were more slender or elongated, especially where the glial fibers were most dense. The predominant nuclear forms were similar in outline to those of astrocytes, as depicted by Penfield in his diagrammatic drawings in his "Cytology and Cellular Pathology of the Nervous System." The nuclei were best studied with ordinary hematoxylin and eosin stains. Many were hyperchromatic, and multinucleated groups were frequently seen, probably the result of cell division (fig. 56). With Mallory's phosphotungstic acid and hematoxylin stain, the fibers were so intensively and selectively stained that outlines of the nuclei were somewhat obscured (fig. 57).



Fig. 50 (case 3).—Transverse section through the nerve and surrounding tumor (removed four and one-half years after the specimen in figure 39). Note the ciliary ganglion at the top of the illustration and the glioma of the optic nerve at the bottom.

The fibers varied in different parts of the nerve. They were coarse and somewhat angulated in some areas, while in others they were more wavy. Some of the fibers appeared to be thick cytoplasmic projections, some running straight for long distances and others being angulated. Small dots, densely stained, appeared in these processes, especially at the terminal ends. These dots in some ways resembled the gliosomes described as normally present in the processes of protoplasmic astrocytes.

The more typical glial fibers penetrated the pial sheath in innumerable places throughout most of the circumference of the nerve (figs. 1 and 58). The pial sheath was somewhat thickened, though separated into layers, between which dense masses of glial fibers had grown. These fibers in places left the nerve in dense sheaves, similar masses often distending the pial lamellae. They penetrated the pia frequently along the borders of the funiculus where the septal prolongations from the pia entered the nerve. They often left the nerve, however, directly through the pia bordering the central area of a funiculus. The sheaves of glial fibers in the pia often appeared without cellular or nuclear attachments, though this may have



Fig. 51 (case 3).—Section of the nerve farther back, toward the apex of the orbit.

been the result of an inability to follow a fiber throughout its course. These glial fibers appeared to run concentrically around the nerve, the dense blue-stained masses alternating with the reddish pink strands of the pia (phosphotungstic acid-hematoxylin stain). They extended just beyond the pia, stopping short of the thickened arachnoid layer beyond, though in one limited area they invaded it a short distance.

The arachnoid sheath was replaced by an enormously thickened mass of tissue made up of proliferating arachnoid cells and some strands of collagen (figs. 1 and 59). These cells resembled, and in places were identical with, the mesothelial cells which

normally line the subdural space and cover the outer aspect of the arachnoid sheath. In one area they assumed an arrangement slightly suggestive of a whorl which surrounds a psammoma body (fig. 60). Other isolated, homogeneously blue-stained bodies had the appearance of typical corpora arenacea (fig 61). Most of the cellular proliferation in the arachnoid, however, appeared in sheets or masses of cells, similar to the meningiotheliomatous type of meningeal tumors described by Bailey and Bucy. They were not divided into lobulated masses, nor did they otherwise resemble the typical dural endotheliomas or meningiomas which are found



Fig. 52 (case 3).—Section through the thickest area of the tumor. Note the marked enlargement of the nerve stem, the proliferation of the arachnoid cells surrounding the nerve stem and the thickened ciliary nerves outside the sclera.

both in the optic nerve sheath and in the brain. Differential stains, particularly Mallory's phosphotungstic acid-hematoxylin stain, brought out in marked contrast the sharp limitation between the glial elements on the one side and the mesoblastic or mesothelial elements on the other, which made up the growth in the arachnoid sheath. The latter mass took a light pink stain with light blue nuclei, while the glial elements took a deep purplish stain. The sharp differentiation of the glial

elements also was clearly shown by other stains, such as Mallory's trichrome stain, aniline blue, Weil's iron hematoxylin, and Van Gieson's stain. The last-mentioned stain was less satisfactory due to the peculiar yellowish brown color it imparts to the normal arachnoid cells. A few cytooid bodies were found in the dense masses of glial fibers within and without the nerve stem. Outside the growth in the arachnoid sheath, the dura, much thickened, surrounded the tumor.

Small blood vessels were seen in the neural portion of the tumor only within the intraseptal spaces. A few small vessels were also present in the area of pro-

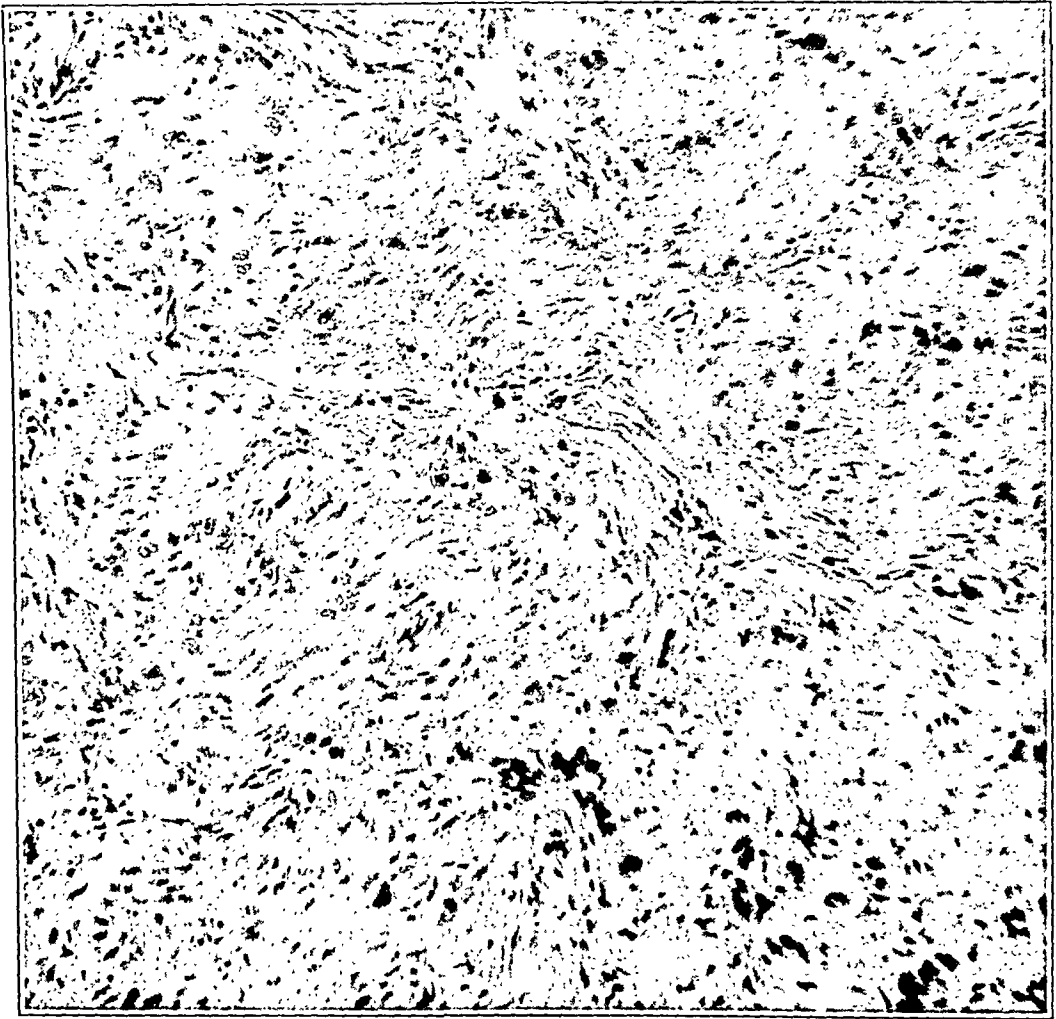


Fig. 53 (case 3).—Transverse section through the posterior half of the nerve (removed four and one-half years after the original operation); hematoxylin and eosin; $\times 200$. Note the enormous increase in glial cells and fibers. (Compare with figure 40, which shows the same nerve when the tumor was in its earliest stage.)

liferated arachnoid. A striking feature of this tumor was the complete absence of vacuolated spaces within the nerve stem. The neuroglial fibers were so numerous and their growth so dense that every vestige of the vacuolated pattern observed in the nerve removed at the original operation had vanished (fig. 57). There were no vacuolated spaces in the thickened tumor in the arachnoid sheath, but the absence

of neuroglial tissue here no doubt accounted for this. As the sections approached the apex of the orbit, the nerve became much smaller and the sheath became thinner. The glial proliferation was unevenly distributed in the nerve stem, one sector showing a massive cellular proliferation, while the remainder of the nerve showed a much milder involvement. This could be seen macroscopically where the nerve was cut at the optic foramen, as previously noted. This uneven distribution of the glial cells extended back into the intracranial portion of the nerve, but was here much less distinct.

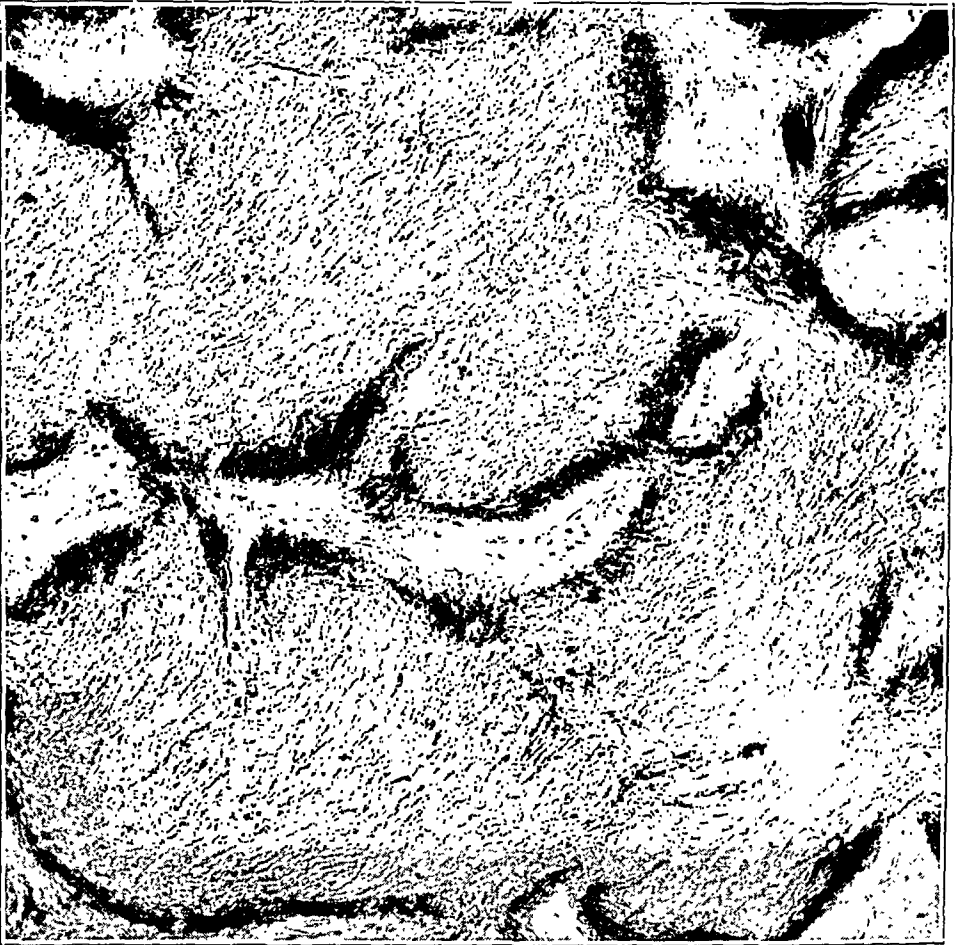


Fig. 54.—Same section as shown in figures 53 and 55 (phosphotungstic acid stain; $\times 200$). Note the marked condensation of glial fibers which border connective tissue septums. The interseptal spaces are dilated, and some contain glial fibers (upper right side).

There were other variations in the sections as one approached the chiasm. The vacuolated spaces again appeared, and fiber formation was less pronounced.

Examination of the Globe and Orbital Contents Outside the Nerve: A complete description of the globe and orbital contents has already been published.⁵

5. Davis, F. A.: Plexiform Neurofibromatosis (Recklinghausen's Disease) of Orbit and Globe: With Associated Glioma of the Optic Nerve and Brain; Report of a Case, *Arch. Ophth.* 22:761 (Nov.) 1939.

Briefly, the study revealed extensive neurofibromatosis of most of the nerves, including the ciliary ganglion. Numerous microscopic tumors were found along the course of the nerves (figs. 62 and 63). They showed varying stages of degeneration, from slight thickening to complete disorganization (fig. 64). Some had progressed to a point of partial liquefaction, such as Antoni described in the reticular type of neurinoma, type B (figs. 64 and 65).

The eye was slightly larger than normal. It showed marked thickening of the uveal tract due to extensive neurofibromatosis of the myelinated and nonmyelinated

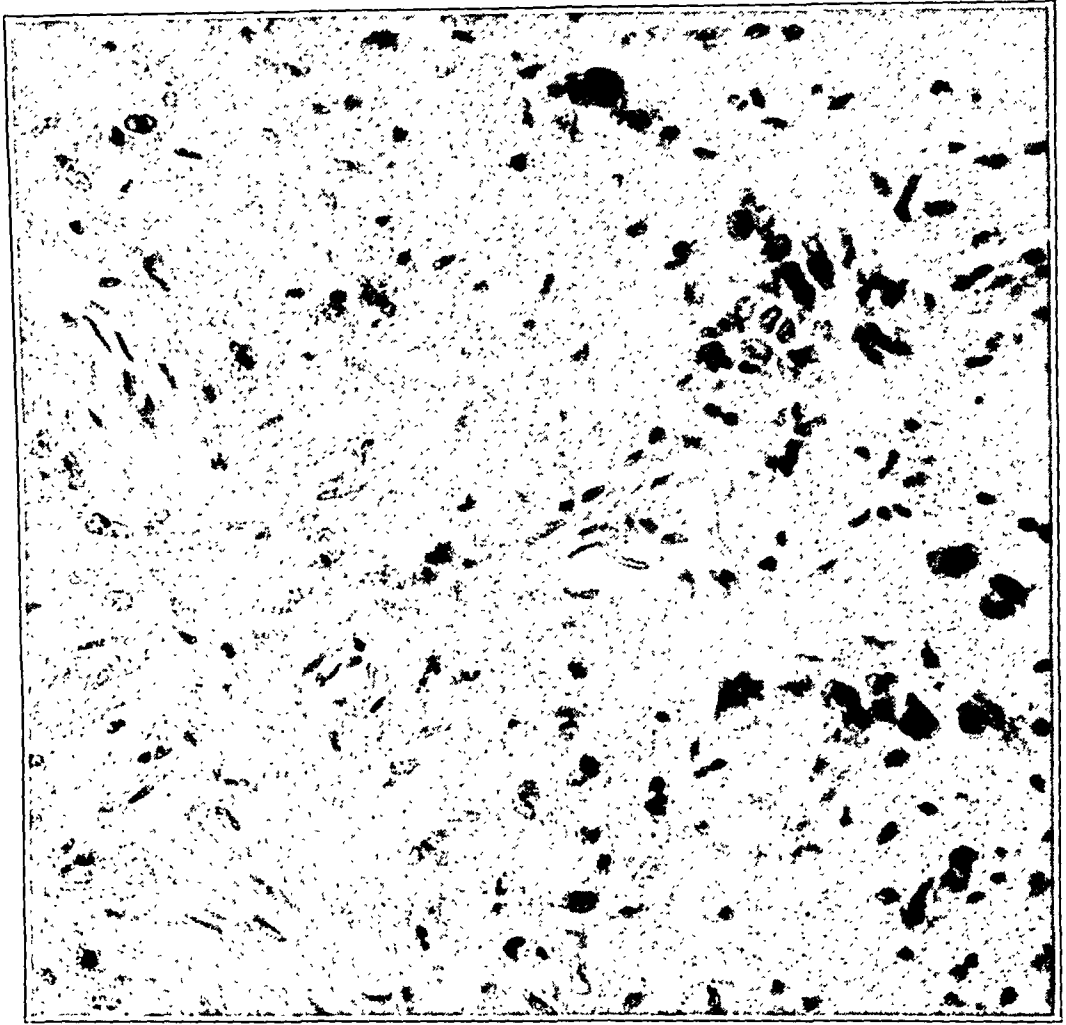


Fig. 55 (case 3).—Same section shown in figure 53 (hematoxylin and eosin; $\times 350$). Note the multinucleated groups of cells. Glial fibers have obliterated the holes noted in figure 41.

nerve fibers (fig. 66). The choroid was about four times its normal thickness in places. The filtration angle was completely obliterated from the adhesion of the thickened iris to the posterior surface of the cornea.

The ciliary nerves throughout the sclera, especially in the posterior segment, were much enlarged due to marked overgrowth of all the supportive elements of the nerves, with degeneration of the myelin sheaths of some of the nerves.

This eye was unquestionably in an early stage of buphthalmos, brought on by blockage of the filtration angle from adhesion of the newly formed tissue of the iris to the posterior surface of the cornea.

Advanced buphthalmos associated with plexiform neurofibromatosis of the lids, orbit and uveal tract is a well recognized, though rare, condition. It is one of the varied manifestations of Recklinghausen's disease.

Diagnosis.—A diagnosis was made of Recklinghausen's disease, manifested by abortive peripheral lesions—café au lait spots; plexiform neurofibromatosis of the orbit; neurofibromatosis of the choroid, ciliary body and iris; glioma of the optic nerve (type, astrocytoma); gliomatosis of the left optic nerve, chiasm, medulla,

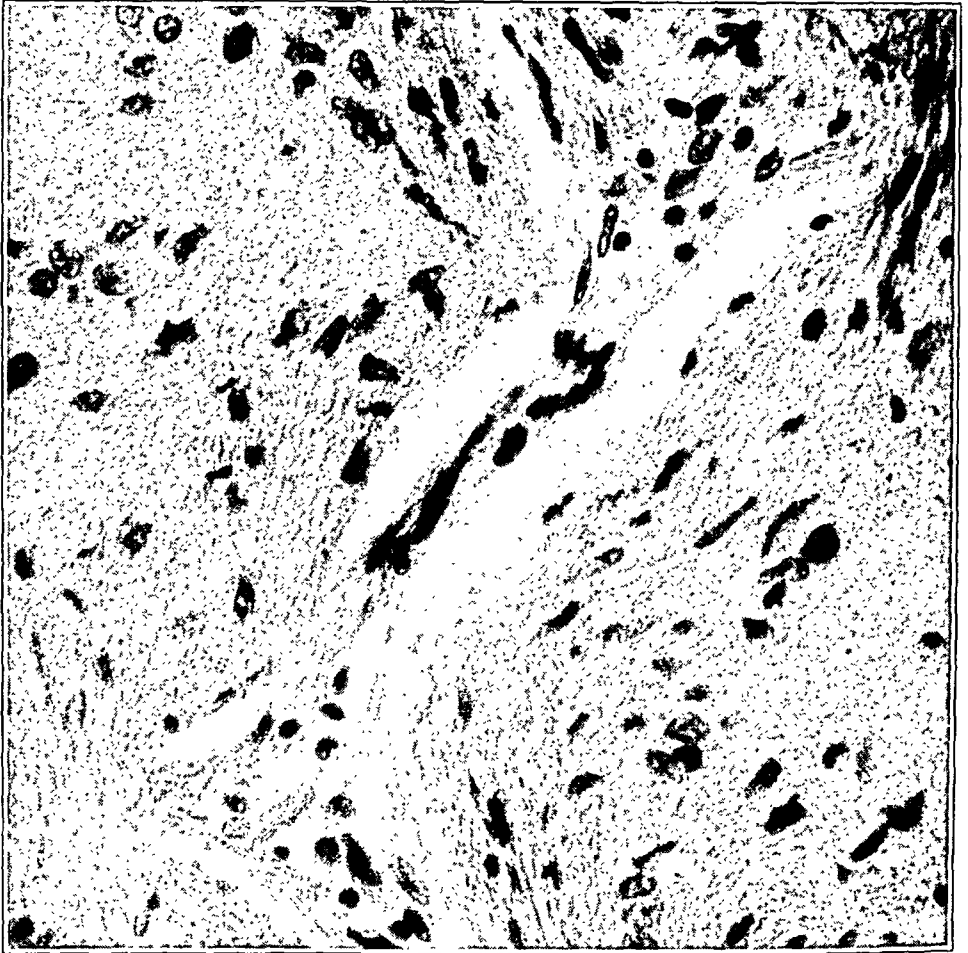


Fig. 56 (case 3).—Transverse section through the tumor of the nerve stem. Same section as in figures 53 and 55. Note the polymorphism of cells. Glial fibers can be seen surrounding nuclei of astrocytes as they pass from one funiculus to another. In these areas and also where the fibers abut the connective tissue septums the nuclei of the astrocytes become more spindle shaped.

pons and thalamus; glioma of the temporal lobe of the brain, and early buphthalmos.

Comment.—The varied aspects of this remarkable case deserve further comment. The early diagnosis was made possible by the fact

that I had but recently studied cases 1 and 2, operation having been performed in the latter case earlier in the same year; otherwise, the slight exophthalmos and pallor of the nerve would scarcely have justified an operation for tumor of the optic nerve. The information gained through the recent study of these cases, however, seemed to warrant exploration.



Fig. 57.—Same section as in figure 54 (phosphotungstic acid and hematoxylin stain; $\times 550$). Note the dense glial fibers throughout the funiculi. They have also invaded the fibrous intraseptal spaces.

I excised part of the nerve, even though it showed no gross evidence of tumor formation and was apparently about of normal size, because of my convictions concerning the diagnosis.

At first no definite tumor formation was noted, although the sections were studied by a general pathologist and by an ophthalmic pathologist.

More extensive study, however, disclosed that early gliosis or gliomatosis was present throughout the nerve. An opportunity was thus afforded for study of these tumors in a very early stage of their growth.

Examination of the nerve at this time and subsequent studies throw some further light on the evolution of these growths. The tumor in this case unquestionably began as an abnormal proliferation of the

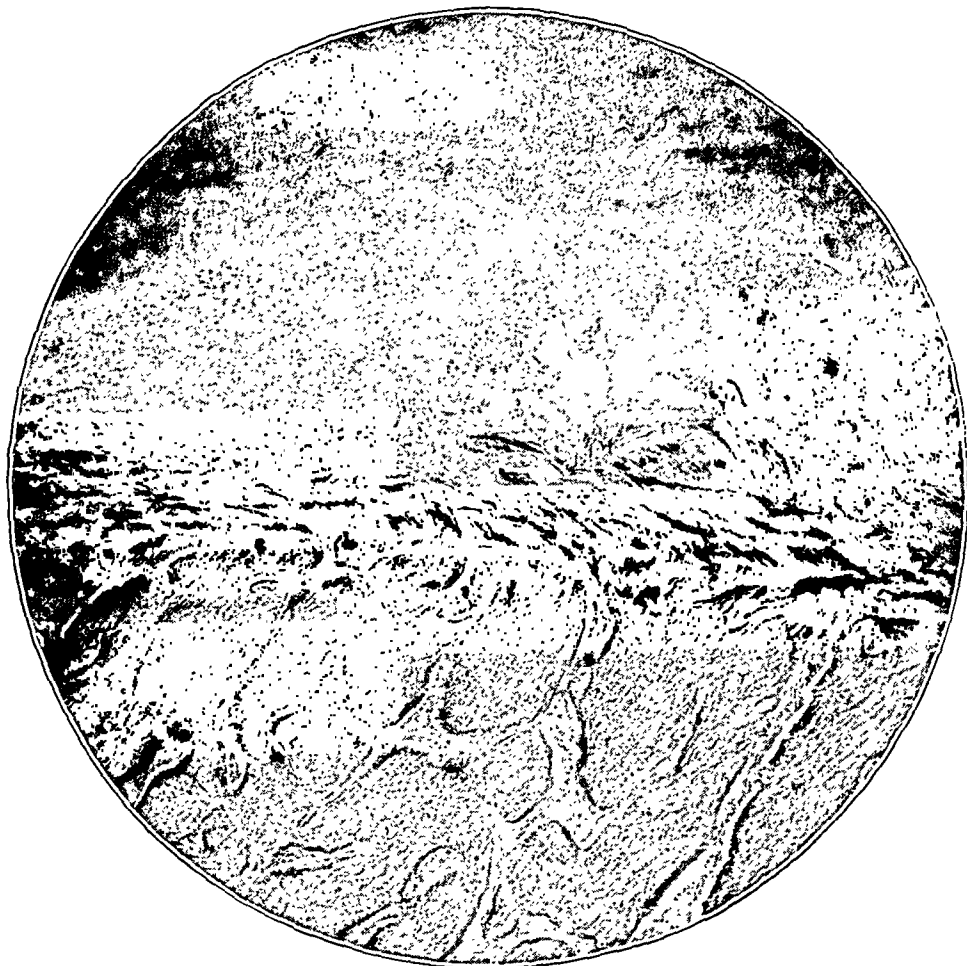


Fig. 58.—Higher power magnification of the section in figure 59. Note the glial fibers invading the pial sheath (the fibers appear black). The proliferating arachnoid cells outside this contain no glial elements. The dura is above.

neuroglial elements of the nerve stem and later spread through the pial sheath into the subarachnoid space. The proliferation of arachnoid cells was sharply defined and of such size that it appeared to be an independent growth, but it was not glial in nature. It seems more likely

that it arose as a process of reaction due to glial invasion through the pia, though it is possible that it arose through some common defect or stimulus.

The marked reduction of visual acuity and the early disappearance of the nerve fibers suggest that some degenerative change in the nerve itself may be the primary change in this disease, while glial proliferation and subsequent tumor formation may be a secondary process.

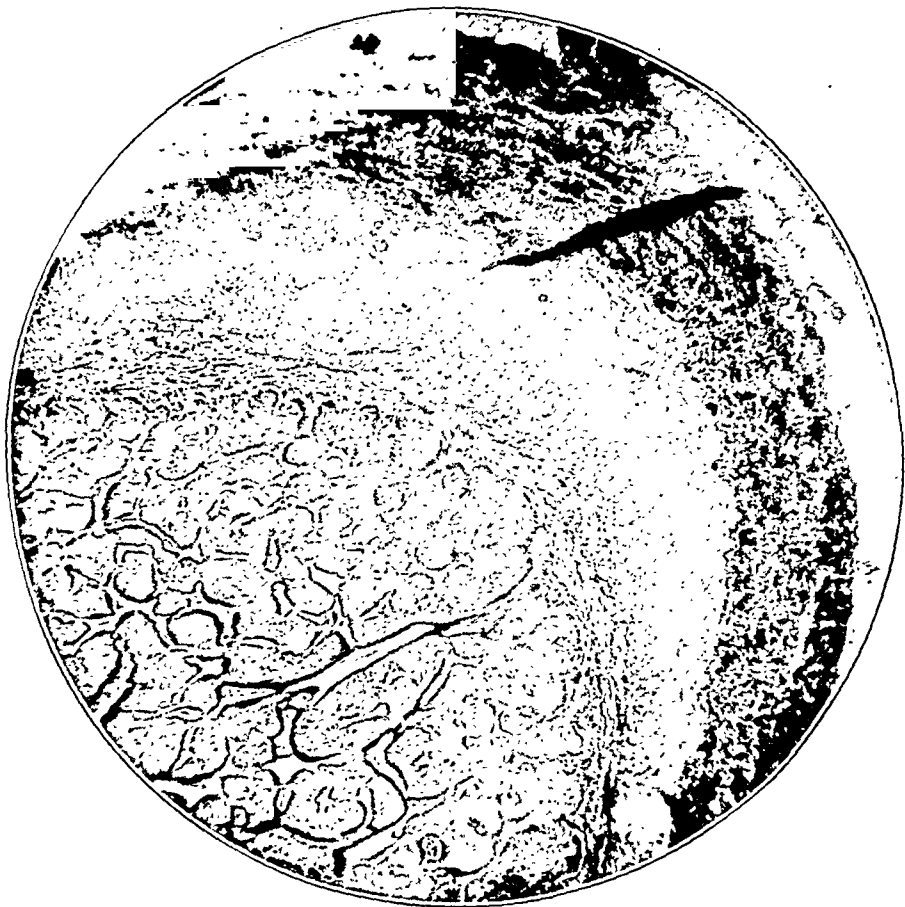


Fig. 59 (case 3).—Transverse section through the tumor (Mallory's phosphotungstic acid-hematoxylin stain). Note the glial fibers in the broken-up pial lamellae. Proliferating arachnoid cells occupy most of the subdural space the same as in figure 1.

Unfortunately only sections stained with hematoxylin and eosin, with Van Gieson's stain, with phosphotungstic acid and hematoxylin and with Weigert's methods were available for study of that portion of the nerve originally removed.

The multiplicity of the lesions, that is, the involvement of the ciliary nerves of the orbit, the nerves of the uveal tract, the gliosis in the

fellow optic nerve, the lesions in the brain and the peripheral changes would seem to furnish abundant evidence that the tumor of the optic nerve was but a part of the syndrome known as Recklinghausen's disease.

The plexiform lesions of the ciliary nerves and the early buphthalmos were not recognized clinically. A slight fulness of the upper lid fold was commented on, but the tumors of the ciliary nerves were mostly



Fig. 60 (case 3).—Section showing an area of proliferated arachnoid cells in the sheath. Note the slight tendency to whorl formation surrounding a psammoma body. This was the only area in the entire tumor, however, in which there was any suggestion of "whorl" formation of cells.

microscopic in size, and no definite masses or cords could be palpated. The marked cupping of the optic nerve was repeatedly noted, but there was no other evidence of intraocular tension. The slight enlargement of the globe was noted, but its real cause was not suspected until the eye was removed. Repeated photographs showed no evidence of an

enlarged globe until the one taken just before the intracranial exploration. There was, to be sure, slight exophthalmos, which persisted after the original operation. This I attributed to possible recurrence of the growth in the unremoved portion of the nerve. It is impossible to know the exact cause. The enlarged globe, the thickened ciliary nerves and the advancing tumor of the optic nerve had all, no doubt, contributed

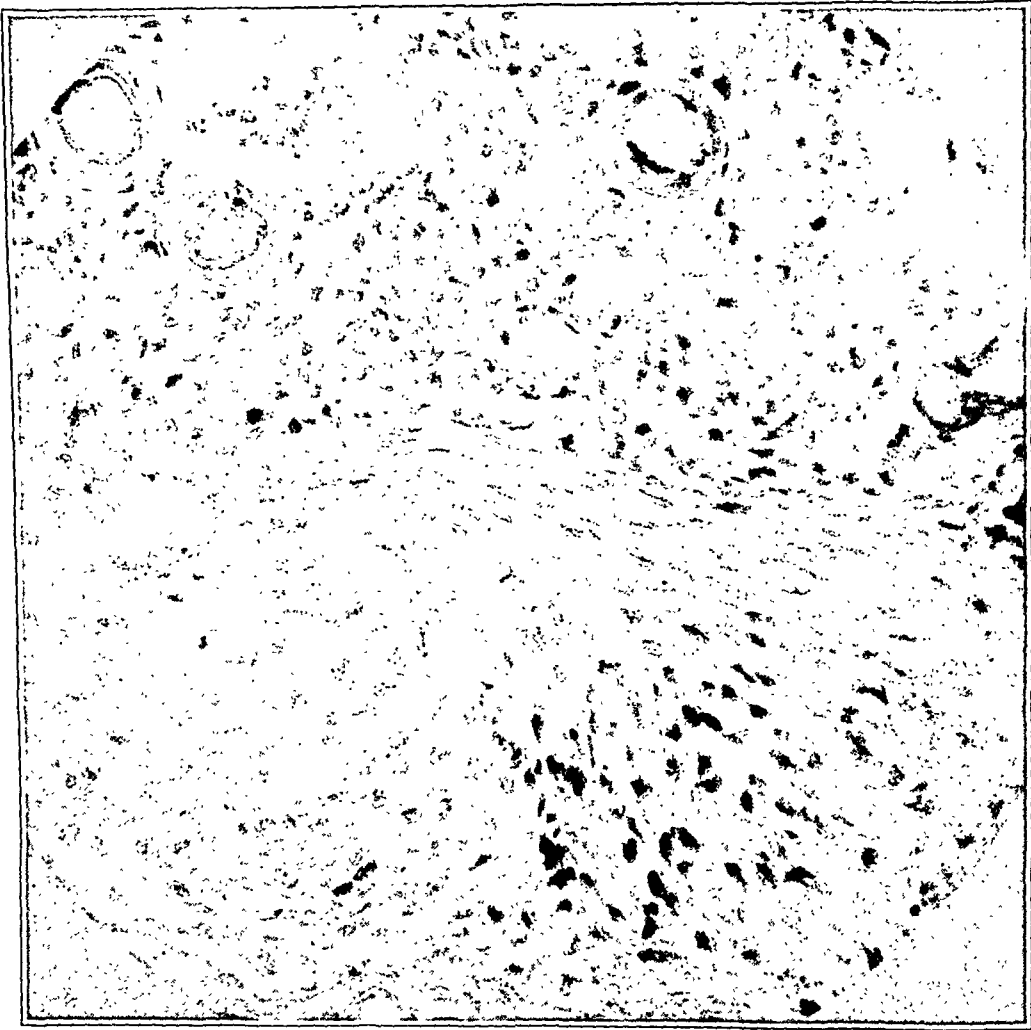


Fig. 61 (case 3).—Detail from the area of the sheath beneath the dura (hematoxylin and eosin; $\times 400$). Note the hyperplasia of arachnoid cells with psammoma bodies.

to it. Viewed in this retrospect, it seems likely that the large and deep cupping of the nerve head was due to early glaucomatous changes, though the cornea was clear and remained so for some years after the sectioning of the nerve, and the intraocular tension never appeared to be elevated. It was taken repeatedly by touch, but owing to the child's lack of cooperation tonometric studies were not made, as this would

have required the use of a general anesthetic. The possible cause of the hydrophthalmos in these cases will be discussed elsewhere.

The roentgen studies in this case have been especially interesting. Numerous examinations were made over several years, which have permitted study of the progressive nature of the lesion. The first films showed no evidence of intracranial pressure, though later ones revealed

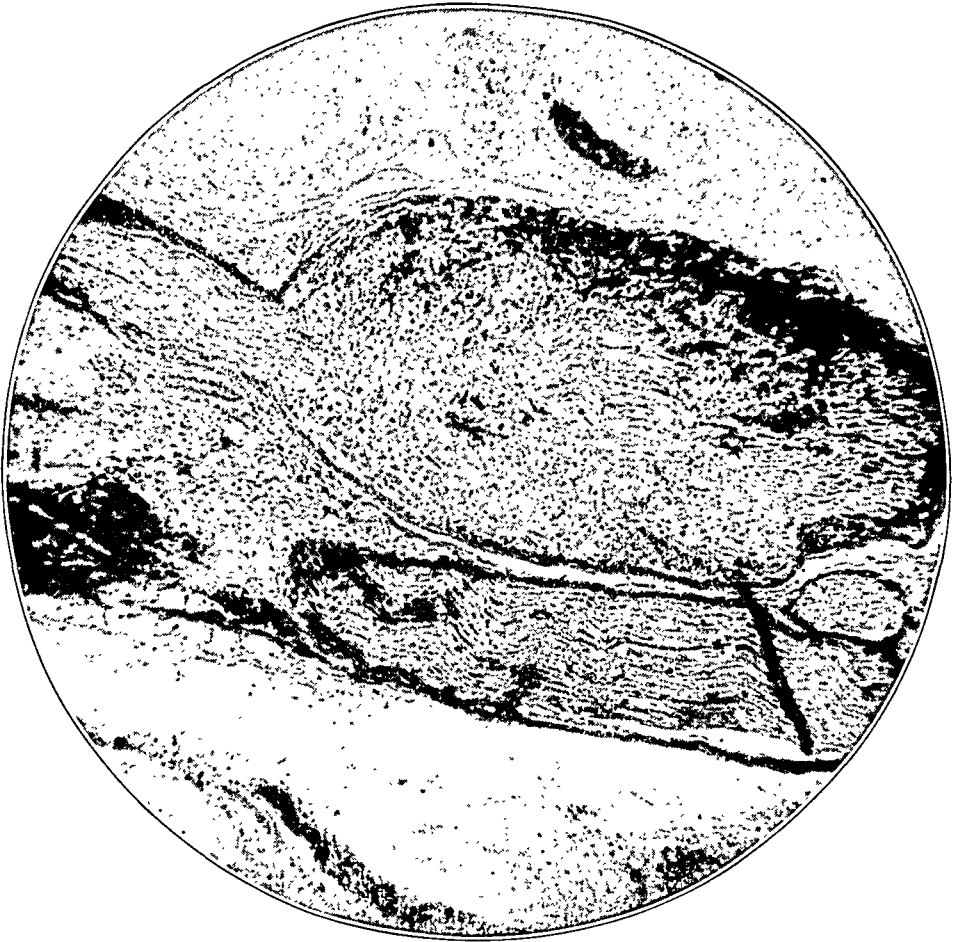


Fig. 62 (case 3).—A tumor developing along the course of a ciliary nerve. Normal nerve fibers are entering the tumor (hematoxylin and eosin stain).

digital impressions. The asymmetry of the orbits was present at the first examination, but its clinical significance was not recognized at the time. It has been reported by others in association with plexiform neurofibromatosis of the lid and orbit. The anterior deformity of the sella turcica was not recognized at the first reading of the films. The first films showed only the slightest sign of it, but the enlargement definitely

progressed, due, no doubt, to the increasing size of the optic nerves and consequent distention of the optic foramina or to erosion under the anterior clinoid processes.

CASE 4 (fig. 67).—*Diagnosis of early glioma of the optic nerve and chiasm, associated with the abortive type of Recklinghausen's disease; cutaneous café au lait-pigmented patches; mollusca fibrosa; transfrontal craniotomy with exploration of the region of the chiasm and optic nerve; attempted unroofing of the right optic canal; cystic glioma of the chiasm and right optic nerve, compressing the left optic nerve.*

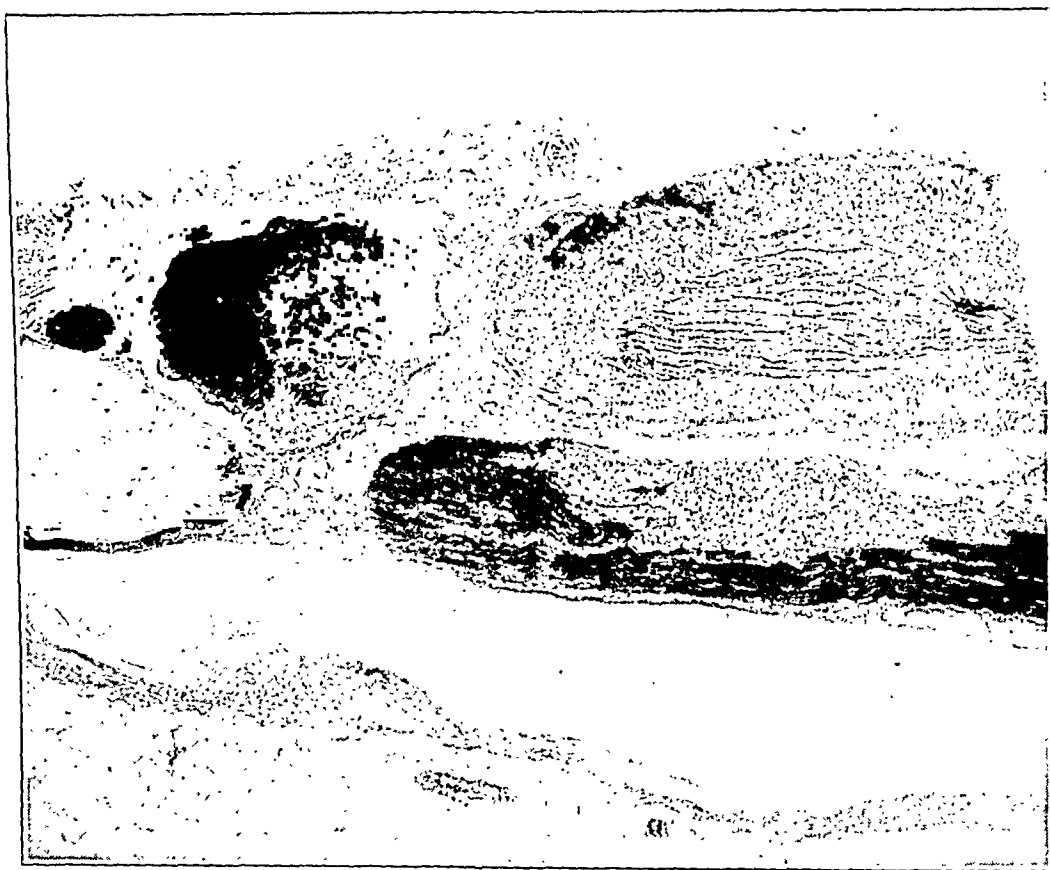


Fig. 63 (case 3).—Ciliary nerves of the orbit (Weigert stain). On the lower right side the nerve fibers have partly degenerated and are replaced by proliferation of fibrous elements of the nerve. On the upper right side a few myelinated fibers remain in the center of the proliferated nerve. On the upper left side a nerve is cut transversely, which is partly degenerated, though most of the fibers are intact. The appearance of the tumors in these nerves suggests a reaction about the nerve which has followed the disappearance of the nerve fibers.

H. L. S., a woman aged 20, was first examined by me on Nov. 30, 1938, in consultation with members of the department of medicine. Her chief complaint was of failing vision and headaches in the back of her head and neck. The patient had been referred to the outpatient service of the department of medicine because of headache and failing vision of three months' duration. She complained of recent

rapid failure of vision in her right eye and of almost daily headaches in the frontal, occipital and vertical regions. The pain in the head was mild and was usually relieved by 5 grains (0.324 Gm.) of acetylsalicylic acid. There were frequent slight nausea and occasional vomiting, which was never projectile in type. The patient's appetite was poor, and her weight had fallen from 158 pounds (71.7 Kg.) to 138 pounds (62.6 Kg.). The remainder of the history was unimportant except for menstrual irregularity, which had existed since onset, at the age of 12. The patient



Fig. 64 (case 3).—Group of nerves of the orbit near the optic nerve. The two smaller nerves show early degeneration. The large nerve at the left has completely degenerated and is jellified or liquefied. This is evidently Antoni's type B "neurinoma."

also complained of nervous irritability, worry and at times melancholia. Physical endurance was reduced, and she felt cold much of the time.

Previous Medical History.—The patient had been admitted to the medical service of the hospital six years previously, when aged 14, for a brief study. At that time she complained of headache, pain in the back of her neck and menstrual irregu-

larity, with nausea and vomiting. She was overweight and had some trouble with acne. A general examination, which included routine laboratory studies and roentgenograms of the skull and sinuses, gave essentially negative results. The sella turcica was normal. Gynecologic examination led to a diagnosis of amenorrhea on an endocrine basis, with no discoverable cause. Nothing definite could be found to account for the headaches. No ocular studies were made at this time, though the patient was subsequently studied by an ophthalmologist. Again, one and a half years prior to the present admission, an ophthalmologist studied her and found

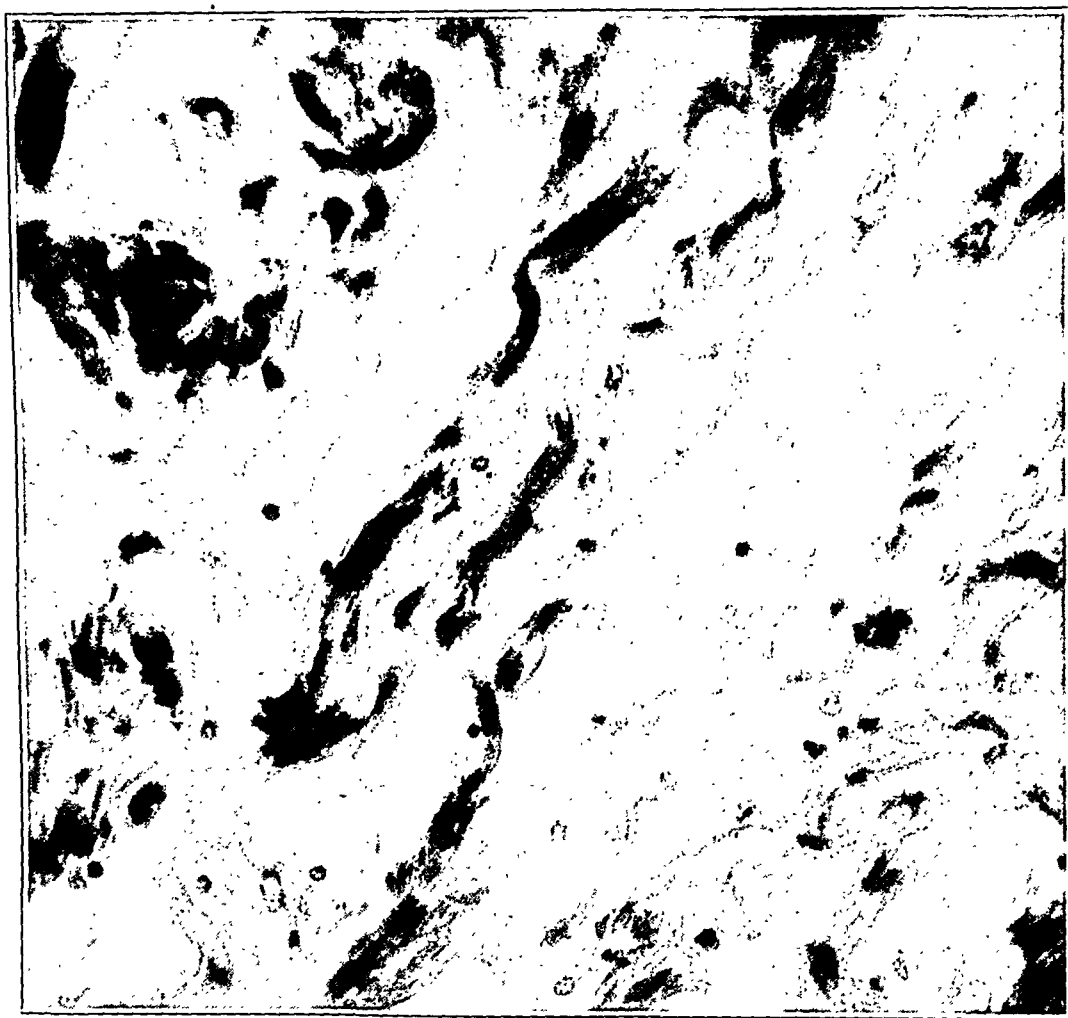


Fig. 65 (case 3).—High power magnification of a tumor similar to that in figure 64. Note the complete liquefaction or jellification of the tumor. Only strands of hyalinized connective tissue remain (Antoni, type B).

only a slight refractive error. She had been recently reexamined by the physician who had previously studied her eyes, and bilateral atrophy of the optic nerve with reduced fields had been noted. Since nothing was found to account for the atrophy, she had been referred to the hospital for study.

Family History.—The family history, obtained on the patient's admission, was irrelevant. Later questioning, however, revealed that the patient's paternal grandmother had lost her vision completely at the age of 78 and, further, that she had

a number of pigmented spots on the skin. The father and mother stated they had never noted any pigmented spots on their bodies, but they were not examined.

Examination of the Eyes.—Both eyes appeared normal to external examination, aside from the faintest widening of the right palpebral fissure. This was so slight that it was easily overlooked on casual inspection. Measurement with the exoph-



Fig. 66 (case 3).—Section through the posterior segment of the globe showing the thickened choroid. The sclera is also much thickened, and the ciliary nerves are much enlarged.

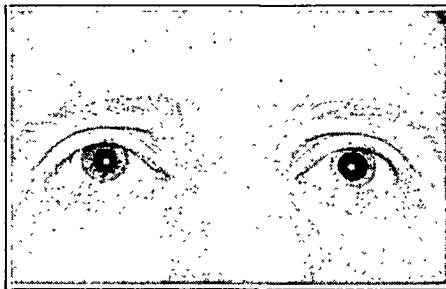


Fig. 67 (case 4).—Glioma of the chiasm and the right optic nerve. The cystic area of the tumor pressed on the left nerve. (Dilatation of the pupil of the left eye was caused by the instillation of homatropine hydrobromide.) There was slight exophthalmos of the right eye, which measured 2 mm. greater than the left eye.

thalmometer confirmed this observation, since there was an exophthalmos of 2 mm. The exophthalmometer reading was 16 mm. for the right eye and 14 mm. for the left eye. Extraocular movements were normal. The pupils were equal and reacted

normally to light and in accommodation. They dilated equally with homatropine hydrobromide. Intraocular tension was normal. Visual acuity was 20/200 in the right eye and 20/15 in the left eye.

Ophthalmoscopic examination showed that the media were clear, and the lens and vitreous appeared normal. There was bilateral atrophy of the optic nerve of about grade 2, the disk of the right eye being somewhat paler than that of the left. The disks were flat, with small central physiologic cupping. The borders were fairly sharply defined, though the superior and inferior margins were less distinct. The left disk showed a thin glial membrane in the physiologic cup which extended over the nasal border for a short distance beyond the disk. There was also a similar opaque, veil-like opacity which obscured the inferior temporal vein at a point about 2 disk diameters from the border of the disk. The retinal vessels in both eyes appeared more narrow than usual for a subject of this age. The veins were slightly tortuous.

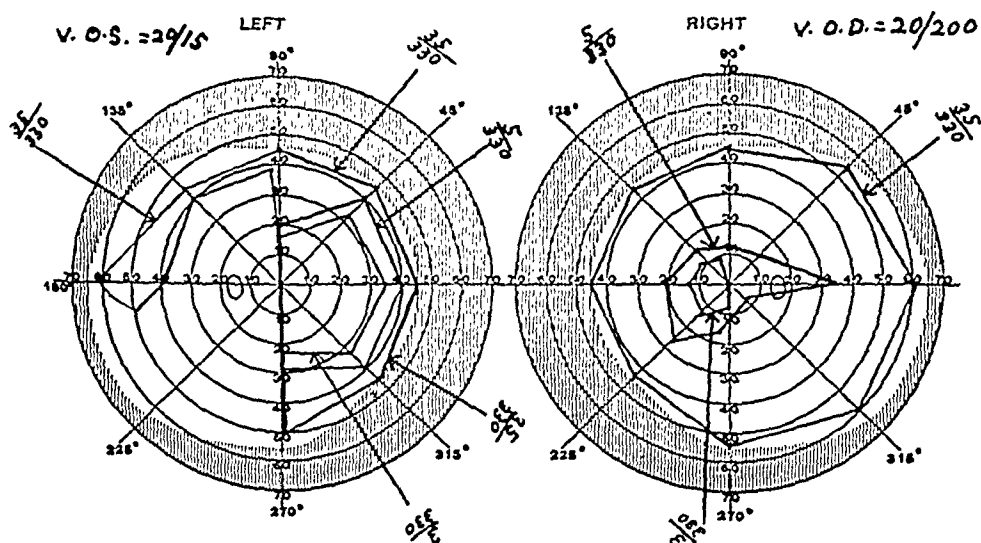


Fig. 68 (case 4).—Visual fields. There was bitemporal hemianopia, somewhat atypical though quite definite, with small test objects.

Study of the visual fields (fig. 68) revealed an atypical bitemporal hemianopia. The field of the left eye for form and colors showed a fairly typical temporal hemianopia with some added constriction in the inferior nasal quadrant. The field of the right eye was much less typical, though hemianopia was revealed with the smaller sized test objects and careful plotting. The extreme peripheral field appeared strikingly normal with a large test object, but the results of repeated tests proved somewhat variable. There was doubtful perception for color. No doubt the extremely poor visual acuity and the marked enlargement of the blindspot interfered with accurate plotting.

General Examination.—The patient was subjected to a complete examination by the department of medicine, including laboratory tests, a Wassermann test, a sugar tolerance test and roentgen examination of the skull, sinuses and optic foramina. In addition, consultations were obtained with members from the departments of surgery, gynecology and neurology. The only findings of importance were a number of light coffee-colored spots in the skin, scattered over various parts of the body. Most of these were typical smooth, nonelevated café au lait patches, such

as are frequently found in Recklinghausen's disease. (Figure 69 shows the location and size of these pigmented areas.) A typical molluscum fibrosum was found on the left breast. It was about $\frac{1}{4}$ inch (0.64 cm.) in diameter, was slightly elevated and felt soft and doughy to the touch. One other similar area was found. The left facial mimic innervation was less pronounced than the right, but voluntary facial movements were normal. There was ringing in both ears, not associated with abnormalities of the cochlear or vestibular portion of the eighth nerve. No cranial pressure points were found. Abdominal and patellar reflexes were slightly

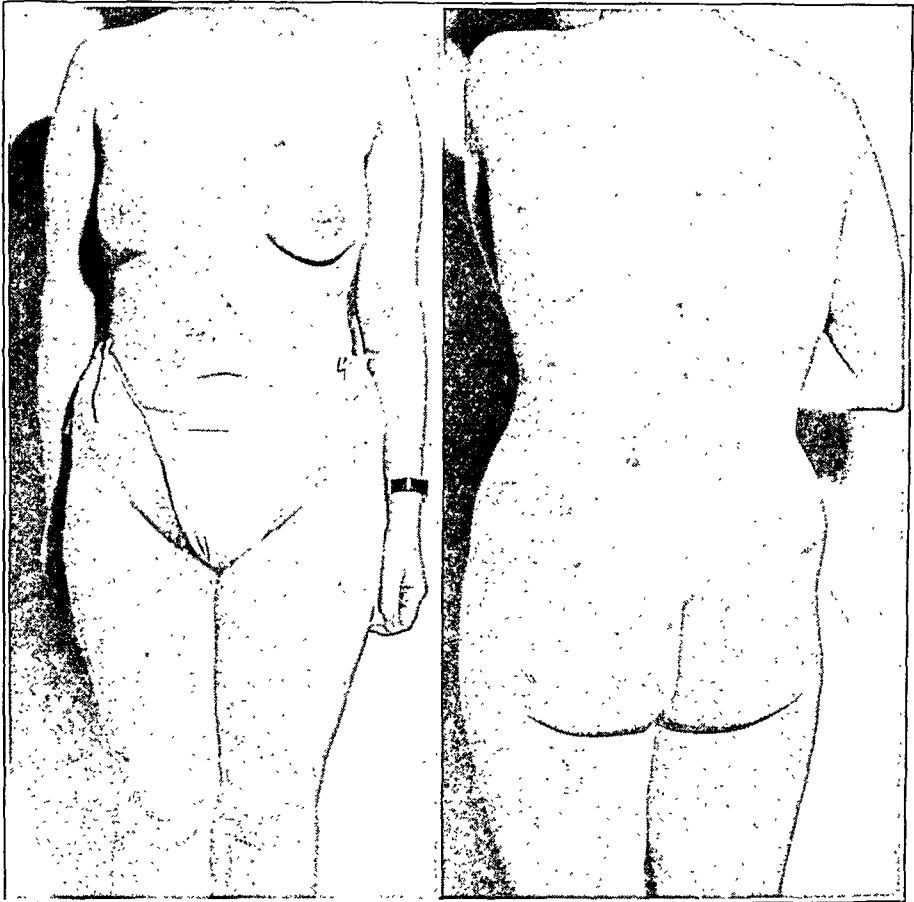


Fig. 69 (case 4).—Front and back views of the patient, showing café au lait-pigmented patches and fibromata mollusca.

decreased. Roentgenograms of the skull revealed larger and more tortuous vessels in the right frontal region than in the left. Lateral stereoscopic views of the skull showed no abnormality of the body of the sella turcica, but a shadow under the anterior clinoid process was suggestive. Views of the optic foramina showed that the right foramen was slightly larger than the left. On the films the right foramen measured 5 by 5 mm. and the left 4 by 4 mm. (fig. 70).

A diagnosis was made of Recklinghausen's disease, with early gliomatosis or glioma of the chiasm extending into the optic nerve. Intracranial exploration

was advised with a view to unroofing the bony foramens and possible incision of the sheaths of the nerves and chiasm.

Operation (Dr. W. Mck. Craig, Jan. 3, 1939).—"With the patient under intra-tracheal anesthesia, a right transfrontal craniotomy was performed, and the right frontal lobe was elevated. The optic nerves and chiasm were easily exposed, and a large mass could be seen within the substance of the right optic nerve (fig. 71). In dissecting the mass free, the left lateral portion was found to be cystic, and when it was punctured a yellowish fluid escaped, allowing the collapse of the mesial portion of the tumor. Because the swelling extended to the optic foramen, an attempt was made to unroof the right optic nerve. This was unsuccessful because of the vascularity of the bone and contiguous tissue. The wound was closed in the usual manner."

The patient made a satisfactory postoperative recovery. A series of high voltage roentgen treatments was advised with the hope of limiting the progress of the growth.

Diagnosis.—A diagnosis was made of cystic glioma of the right optic nerve and chiasm.



Fig. 70 (case 4).—Roentgenograms of optic foramens (actual size). Note that the foramen on the right side is slightly enlarged.

Comment.—Since the preoperative diagnosis in this case indicated a lesion of the chiasm as well as of the optic nerve, operation was advised, chiefly with a view to decompression of the nerves by removal of the roof of the bony foramens. Further, since vision was rapidly failing and blindness appeared inevitable, exploration seemed warranted.

While it was not considered wise to jeopardize the vision by removal of a section of the tumor for biopsy, there is no question that pathologic examination in this case would reveal a gliomatosis similar to that noted in the chiasm in case 3. The tumor had evidently progressed to a somewhat more advanced stage than the chiasmal lesion in case 3, since the chiasm appeared enlarged. The right nerve also appeared to be approximately twice its normal size, but the left nerve, to gross inspection, was not definitely enlarged.

The pigmented patches in the skin, with two small mollusca fibrosa, together with the atrophy of the optic nerve and a fairly typical chiasmal field change, made the diagnosis simple.

The small glial veil bordering the disk may be an early gliosis of the nerve head. The area to the inferior temporal side may also be of the same nature. A glioma of the neuroglial elements of the retina may occur, producing a true glioma of the retina, which is not to be confused with retinoblastoma or so-called glioma retinae. A tumor of this nature (astrocytoma) has been reported by McLean (1937). Later progression in these areas may throw further light on this subject.

This case again strikingly illustrates the diagnostic value of the classic signs of the mild, abortive type of Recklinghausen's disease. At

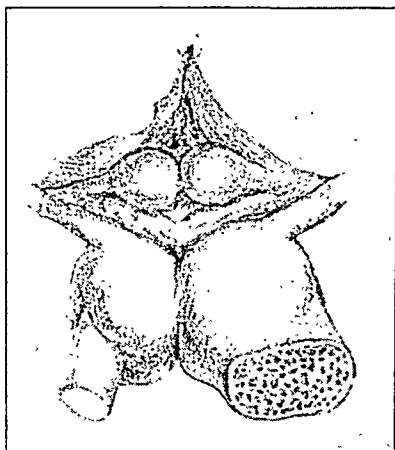


Fig. 71.—Section from a tumor described by Michel in 1873, which was also associated with Recklinghausen's disease. The specimen was similar to that found in case 4 at operation.

the original routine examination of the patient a note was made that "several cloasmas were present in the skin," but they were not considered as having any bearing on the diagnosis. Apparently general physicians have attached little or no importance to these signs, since their relation to lesions of the central nervous system which sometimes accompany them has not received general recognition.

It is my hope that this study, together with the reports cited in the literature, may in a measure aid in bringing about a better understanding of the significance of these phenomena and their importance as a diagnostic aid when associated with atrophy of the optic nerve.

CASE 5 (fig. 72).—*Diagnosis of gliosis of the optic nerves and chiasm, associated with the abortive type of Recklinghausen's disease, cutaneous café au lait-pigmented*

patches and subcutaneous neurofibromas; transfrontal craniotomy with exploration of the chiasm and optic nerves and unroofing of the right optic canal, extending into the orbital plate, followed by incision through the sheath of the optic nerve; enlargement of both optic nerves extending to the chiasm.

Mrs. R. F., a white woman aged 26, was admitted to the ophthalmic department of the Wisconsin General Hospital on Feb. 13, 1939. Her chief complaint was failing vision and headaches.

History of Present Illness.—The patient stated that her vision had been poor as long as she could remember. She recalled that she was unable to see the black-board while sitting in the classroom when she was in grade school. She was



Fig. 72 (case 5).—Gliomatosis of both optic nerves. Note the slight bilateral exophthalmos and subcutaneous tumors of the face and cheek.

fitted with glasses at the age of 10 but noted no appreciable improvement in vision. The status of her vision remained about the same until April 1936, when it grew worse after the birth of a baby. The vision of the right eye then failed progressively for about two years, when it became stationary, while that of the left had grown gradually worse up to the time of the patient's admission. Corrected vision at that time was 20/200 in the right eye and 1/200 in the left eye. There was no history of diplopia or of inflammatory symptoms referable to the eyes. The patient complained of fairly severe headaches which were almost a daily occurrence. The headaches were worse in the morning and gradually subsided about the middle of the day. She was not nauseated at any time. She gave a history of some menstrual disturbance. There were metrorrhagia and menorrhagia.

Questioning of the patient revealed that she had always had peculiar spots on her skin (fig. 73), and she believed that these had become larger within the last few years. Cutaneous nodules had been present for about five years, and one situated beneath the surface of the skin or on the dorsal aspect of the left metacarpal bone of the thumb had been present since birth.

Previous Medical History.—In 1928 the patient suffered an injury to the hip while working in a hay field and had since had a definite left foot drop and limps with the left leg. The history aside from that related was unimportant.



Fig. 73 (case 5).—Café au lait-pigmented patches in the skin of the trunk and extremities.

Family History.—The patient stated that so far as she knew no member of her family had any serious disturbance of vision or any pigmented spots on their bodies. She made a similar statement regarding her two children, but subsequent examination revealed that 1 child, a girl of 3 years, had numerous pigmented patches in the skin distributed over various parts of her body.

Examination of Eyes.—External examination of the patient's eyes showed them to be in normal position. Extraocular movements were normal except for a slight limitation in the field of the left external rectus muscle. There was an

unsustained nystagmus when the patient looked to the right. Both eyes seemed to be somewhat prominent, and exophthalmometer measurements were 15 mm. for the right eye and 17 mm. for the left eye. There was a definite widening of the palpebral fissures which produced a starey appearance (fig. 72 *A*). The pupils were equal and reacted normally to light and in accommodation.

Examination of the right eye showed a normal anterior segment. The vitreous body was clear. The nerve head showed rather advanced atrophy with a physiologic excavation and a piling up of a moderate amount of pigment about the margins of the disk. The vascular system showed moderate narrowing of the arteries and a slightly increased fulness of the veins. There were no hemorrhages. There was an occasional small deposit of pigment about the periphery of the retina. The retina itself showed numerous small areas out toward the periphery that appeared to be grayish streaks. These suggested the possibility of some glial proliferation. The macula was normal.

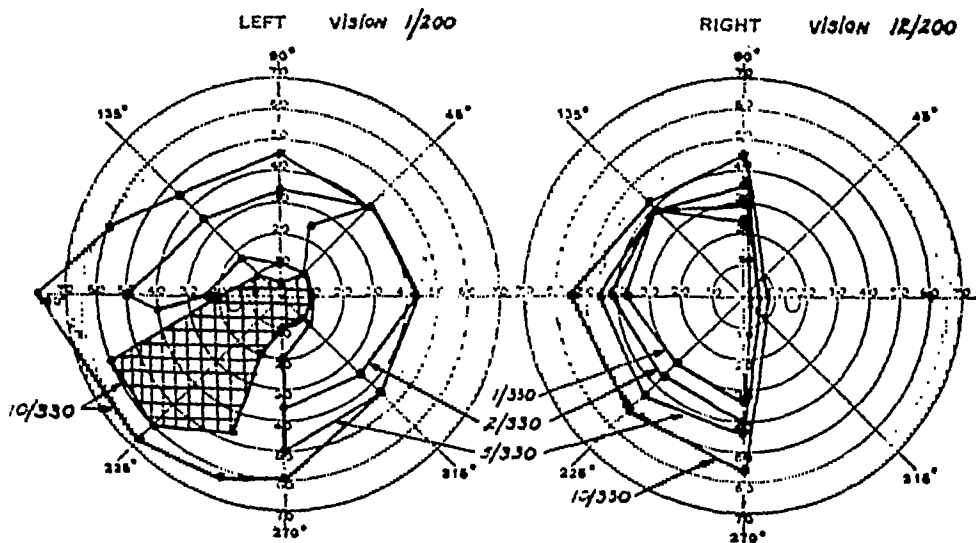


Fig. 74 (case 5).—Visual fields showing bitemporal hemianopia.

External examination of the left eye also gave negative results. The appearance of the fundus was much the same as that described for the right eye, with the exception that the atrophy of the optic nerve in this eye was much more advanced. The tension in each eye was 13 mm. (Schiotz). There was no pain on pressure over the eyeballs, but there was some pain when mild pressure was exerted over the lateral rims of each orbit. There was no fulness in the soft parts of either orbit.

Careful studies of the visual fields with a 3 mm. target revealed a bitemporal hemianopia (fig. 74). There were a central scotoma in the left field and complete loss of color perception. Visual fields obtained with various-sized targets are also shown in figure 74.

General Examination.—As far as the primary condition was concerned, the results of the general examination were essentially negative with the exception that over the entire surface of the body there were many café au lait-pigmented

spots. These varied considerably in size. There were many subcutaneous nodules, particularly over the forearms and hands.

The blood pressure was 100 systolic and 60 diastolic.

Roentgenograms of the optic foramina revealed slight bilateral enlargement, the left being larger than the right. Roentgenograms of the skull were found to be essentially normal. The sella turcica was normal in size and contour.

Laboratory examination showed nothing of especial interest. The blood chemistry was within normal limits, and the Wassermann reaction of the blood was negative.

An audiogram showed the patient to have a 15 per cent loss of hearing in the right ear and a 26 per cent loss in the left. No clinical significance was attached to this finding.

The patient was seen in consultation by Dr. H. H. Reese, of the neurologic department, who reported the following positive findings: There was a certain euphoric attitude out of proportion to the patient's disability. There was an evident facial asymmetry, a difference in the two arms and some slight alteration in the pelvis and legs, the left leg being the larger. Examination of the cranial nerves revealed weakness of the entire left seventh nerve, more marked in the oral branches. There was mild paresis of the abducens nerve on the left side. The motor strength was equal except for mild paresis of the left peroneal nerve. There was discoordination in the adiokokinetic phenomena, with suggestion of rebound phenomena, and in the performance of hopping tests, the left leg being less efficient. Sensation for all modalities was found to be intact. Reflexes revealed a hyperactivity on the left side with a well established Babinski sign. It was the opinion of Dr. Reese that the multiplicity of findings could not be conditioned by a single lesion. He felt that there was evidence to warrant suspicion of a lesion in the right frontobasal and the left cerebellar region, plus the evident lesion in the region of the chiasm.

The patient was also seen in consultation with Dr. Robert Burns, of the orthopedic department, who found that the gait was unsteady. The left lower extremity was spastic. The left hip was high. When the patient was standing at ease, the weight was borne chiefly on the right lower extremity. There was total scoliosis with convexity to the right, which was obliterated by forward bending. Roentgenograms of the spine, of all the bones of both legs and of the pelvis revealed only the presence of scoliosis in the lumbar region with convexity to the right and small elongated cystic areas in the lower ends of both fibulas. These could not be definitely said to be evidence of involvement by neurofibromas.

A diagnosis was made of gliosis or glioma of the chiasm with extension into the optic nerves. Other lesions within the brain were considered as a distinct possibility.

Transfrontal exploration was advised, with a view to decompression of the optic nerves and possible incision of the sheaths of the nerves.

The patient was under observation for about two weeks, since she had a mild secondary anemia.

Operation (Dr. E. R. Schmidt; March 7, 1939).—Transfrontal craniotomy was performed. Exploration was carried out first on the left side, and the optic nerve was easily exposed. It was found to be more than twice its normal size,

and the sheath was smooth and tense. Similar exploration followed on the right side, and the optic nerve was found to be fully as large as the left. The roof of the bony canal and a part of the orbital plate were removed on the right side, following which an incision about $\frac{3}{4}$ inch (1.9 cm.) in length was made in the sheath of the nerve. The enlarged nerves could not be satisfactorily exposed where they joined the chiasm. Attempts to elevate the frontal lobes sufficiently to visualize the chiasm were attended by bleeding, so it was deemed unwise to proceed further in this direction. No effort was made to uncap the left nerve because of the almost total loss of vision in this eye and the added time which would have been required for this procedure.

The patient made a prompt and satisfactory postoperative recovery. No change was noted in her vision one week after operation.

Diagnosis.—A diagnosis was made of gliosis of the optic nerves and chiasm.

Comment.—The similarity of the physical findings in this case and in the preceding one is evident. That these 2 patients sought consultation about the same time is remarkable when one considers the rarity of these tumors. Since in neither case was roentgen evidence found of disease within the sella turcica, each might have been passed over for an obscure case of atrophy of the optic nerve due to retrobulbar neuritis. With the presence of the telltale signs of Recklinghausen's disease, however, as noted in the coffee-colored spots in the skin and the presence of the subcutaneous tumors, one should not long remain in doubt concerning the diagnosis.

Cushing, in his classic paper concerning the chiasmal syndrome, has so clearly presented the important features of these cases that any one who reads it should be able to make a diagnosis. Gliomas which involve the chiasm and intracranial portion of the optic nerves are less common than the intraorbital variety, as pointed out by this author. Exophthalmos is usually absent, at least for a time, unless the growth extends into, or later develops within, the orbital part of the nerve.

Changes in the visual fields of these cases are indispensable for diagnosis. The hemianopia in the less affected eye was sharply limited in this case, which, according to Cushing, is somewhat contrary to the rule. The extremely poor visual acuity in the left eye made it difficult to obtain a perfectly accurate and uniform field.

Undoubtedly the lesion of the optic nerves also involved the chiasm. The difficulties encountered in making a satisfactory exposure of the chiasm may have been due to its "post-fixed" position or possibly to the diffuse invasion of the surrounding region which prevented elevation of the frontal lobes.

(To Be Concluded)

IMPRESSION TECHNIC FOR CONTACT GLASSES

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The newer method of fitting contact glasses by a mold has superseded the previous trial and error method. The stock lenses will fit in about 25 per cent of cases. There are so many variations in the shape and size of the sclera and cornea that it is impossible for all eyes to fall into certain classifications of sizes. Perhaps in the future a testing set containing many sizes and shapes will suffice, but at present the impression and mold method is preferable.

The more impressions I make of eyes for contact glasses, the more I am convinced that a glass made from a mold of an eye is superior to a stock lens; also, that in order to have a good molded glass, it is necessary to have as good an impression as it is possible to make. The better the impression, the better the glass will fit. If the impression is not perfect, one cannot expect the manufacturer to make a good glass. Furthermore, if a technic is used which will enable changes to be made in the glasses, work with contact glasses will be more successful.

In making an impression of the cornea and the anterior part of the sclera, accuracy of the impression is of paramount importance. At best, it is possible to obtain only a poor approximation of the eye. Factors which hinder the production of a perfect negative form are: the pressure used in placing the impression material, an imperfect plastic, movement of the eye and the fact that soft, pliable tissue is being worked with rather than a solid surface.

The technic which I find gives the best scleral approximation is with the use of dental wax, such as Kerr's dental wax of gage 20. The old negocoll-hominit procedure is still the best for an impression of the cornea and the limbus.

Dental wax of gage 20 is 0.7 mm. thick. The first step is to determine the approximate scleral curve by means of a round glass of known curvature. A piece of wax 3 inches (7.5 cm.) square is cut. It is slowly molded over a hemisphere of the same radius of curvature as the sclera of the eye. Different sizes of hemispheres are used, such as 11, 11.5, 12, 12.5 and 13 mm. It is best to start with the largest size and mold the

This paper was presented as a graduate lecture at the Forty-Fourth Annual Convention of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 10, 1939.

wax on successively smaller hemispheres until the same radius of curvature as that of the eye is obtained. The center of the wax is then punched out with a punch 11 mm. in diameter. A glass center 12 mm. in diameter is placed over the hole and cemented with wax. Next, the overall dimensions are determined by inserting different sizes of contact glasses in the eye. I use those 20 by 22 mm., 22 by 24 mm. and 24 by 26 mm. in size. A contact glass of the chosen size is placed over the wax form, and the wax is cut around the edge of the glass. A wax form is now available with a glass center of the approximate scleral curvature of the eye and the dimensions of the desired glass. This is placed in ice water for five minutes. It is then put in the anesthetized eye for fifteen minutes, care being taken that the center of the glass is over the center of the cornea. The eye should be fixed in one position as nearly as possible for fifteen minutes. The form is then sprayed with ice water to harden it. A rubber sucker is applied to the glass center, care being taken that in removal the form does not touch the lids. If it does, the impression will be incorrect. The form is placed in ice water, and preparations are undertaken to make the positive form.

This is made of dental stone. By practice, one can learn the proper proportions of powdered stone and water to be used. The mixture of stone is placed in the wax mold; an additional stone mixture is placed into a mold, and the wax is inverted over the mold filled with stone until it hardens. After it is hard, the wax is trimmed away and the positive form is completed.

A skeleton glass is made from this form. Often the glasses as received from the manufacturer are not a perfect fit, even though they may be a close approximation of the cast of the eye. The reason for this is the impossibility of making an exact impression of an eye, as stated previously. Therefore, it is well to use a glass which can be corrected or, if necessary, to have a new glass made which will incorporate any necessary changes. The fitting of the glass to the eye is the difficult part of contact glass work, and it requires much time and patience, both on the part of the examiner and on the part of the patient. It is better to make the first observation of a glass without any solution in the anesthetized eye. Various points are noted. The overall size is of first importance. It must be large enough to cover a wide area of sclera without going into the lower fornix or onto the semilunar fold. The convexity and depth of the glass are noted. A glass should be just deep enough to clear the cornea. Naturally, it must not rub over the cornea, and it must not be too deep or there will be a sliding of the glass over other parts of the cornea or limbus when the eye is moved. Finally, the limbic area is watched for pressure and sliding. The glass is removed, and 3 drops of fluorescein is placed in the bowl, which is then filled

with saline solution and reinserted. Now, areas of contact will be shown, and the glass should be looked at with the slit lamp. If the glass contacts at forbidden areas, these places are marked on the glass and on a plaster of paris copy of the glass and returned to the manufacturer for correction.

If the glass looks good in the eye, it is removed and reinserted with physiologic solution of sodium chloride and worn for a four hour testing period. If the glass can be worn comfortably for two four hour periods without causing any corneal abrasions or air bubbles, it is ready for optical grinding. If it causes burning, tearing or redness, obviously the fit is incorrect. The formation of air bubbles is a frequent and annoying occurrence. They are due to a glass being of too great or too little convexity. An aid in finding the point of entrance of air is to place a drop of fluorescein at the edge of the glass and to watch its entrance into the bowl of the glass. A yellow channel may be seen, indicating the point of entrance of air.

When it is determined that the skeleton glass is correct, a finished glass is made with the proper optical correction. To arrive at the proper corneal prescription, it is necessary to have only a few afocal test glasses of known radius of curvature, such as 8, 8.5 and 9 mm. Ordinary lenses from the trial case can then be added in a trial frame to obtain the desired vision, and the distance between the contact glass and the auxiliary lens measured.

The best solution to use is physiologic solution of sodium chloride buffered with sodium bicarbonate. After considerable experimental work, I have come to the conclusion that there is no value in having a solution which will conform to the p_H index of the patients' tears. If a contact glass causes redness, tearing, or discomfort, it is because it is ill fitting, and although a solution of a different p_H index might give temporary relief, there can be no lasting benefit. If a contact glass causes untoward reactions, there is probably pressure at the limbus or on the cornea.

Requisitions for a good fitting contact glass are the following: It must be the proper size so that it covers a large area without any pressure on the peripheral portion. It must not be in contact with the cornea or limbus. It must not ride or rub over the cornea or limbus when the eye is moved. Air bubbles must not form under the glass. The glass must not cause the formation of corneal abrasions. It must be worn for four hour trial periods with comfort on two successive days. The optical correction must give the desired vision. Only when these requisites are fulfilled can a contact glass be called satisfactory.

VORTEX-SHAPED DYSTROPHY OF THE CORNEA

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In 1910 Fleischer¹ described the eyes of a 37 year old woman suffering from multiple sclerosis in an article entitled "A Peculiar Previously Unknown Opacity of the Cornea." The left amblyopic eye of this patient showed convergent strabismus. Refraction of both eyes revealed high hyperopia with considerable astigmatism at an oblique axis. The corneas showed identical changes. In the superficial layers there was a fine opacity covering the whole cornea. It consisted of rays or bundles converging toward a point slightly below the center of the cornea or radiating from that point toward the periphery, like a crown of human hair. The lower and upper halves of the cornea were separated by a narrow horizontal stripe of clear corneal tissue. Higher magnification showed that the opacity consisted of small yellowish brown points and spots and that the picture of the vortex figures was caused by clear spaces between these spots. The surface of the cornea was clear and smooth. One and a half years later the condition was not changed.

In 1930 Lutz² reported a similar condition in a mother and her daughter. A 26 year old woman came under his observation with acute conjunctival inflammation due to hair dye. A fine, almost identical corneal opacity was observed in each eye which consisted of stripes of yellowish brown spots, converging toward the center of the cornea, where they formed a whirlpool-like figure. These stripes were broader on the periphery than in the center; they gave a scalloped appearance and were partly interrupted by clear tissue. Occasionally two or three peripheral stripes joined into one. Lutz located the opacity on Bowman's membrane or in the superficial layers of the substantia propria in a plane parallel to the surface. The vision in both eyes was 5/6. The Javal ophthalmometer showed 1.5 D. of astigmatism. The refraction was not given. The corneal sensibility was normal. The 53 year old mother of this patient showed the same changes in her corneas. After correction of hyperopia of +2 D., vision was normal in both eyes; otherwise the eyes showed no pathologic changes.

From the service of Dr. Webb W. Weeks, Beth Israel Hospital.

1. Fleischer, B.: Arch. f. Ophth. **77**:136, 1910.

2. Lutz, A.: Arch. f. Ophth. **123**:704, 1930.

In 1931 Vogels³ contributed an additional case. A 60 year old woman had a veil-like opacity in both eyes. Slit lamp examination proved the corneal surface to be smooth and shiny. Fine yellowish brown particles formed a whirlpool-like or vortex-like figure, terminating or beginning slightly eccentrically. The whirlpool in the right cornea was more temporally from the center and in the middle line. Fine radial stripes started from this center and widened toward the corneal margin, similarly to the tail of a comet. Between these stripes was a zone of clear corneal tissue.

REPORT OF A CASE

M. G., a 16 year old white girl, had had the usual diseases of childhood and had been seriously ill with infectious mononucleosis two years before the present examination. She had been wearing glasses for the past ten years, the



Fig. 1.—Appearance of the right (A) and left (B) corneas.

last change having been made one year previously. The family history did not reveal any malformation or any hereditary disease of the eyes. The general examination gave negative results.

Ocular examination showed the palpebral fissures of both eyes to be equal. The lids were normal. There was no exophthalmos or enophthalmos. The ocular movements were normal, and there was no nystagmus. The conjunctiva of each eye was not injected, and there was no ciliary injection. Focal illumination revealed a fine opacity in each cornea. Irregular stripes of opaque corneal tissue started on the periphery and joined in a whirlpool slightly below and nasally from the middle of the cornea. Slit lamp examination showed that these opacities consisted of yellowish brown spots. They were located in the deepest layers of the epithelium and could not be seen underneath Bowman's membrane with the highest magnifications. The stripes were separated by channels of absolutely clear corneal tissue. The deeper layers of the cornea were normal. There were no keratic precipitates. The corneal sensitivity was normal. The irides were brown and of normal structure.

3. Vogels, A.: *Klin. Monatsbl. f. Augenh.* **86**:591, 1931.

The pupils were round and equal and reacted to light and in convergence. The lenses did not show any opacities. The media were clear. The fundi showed marked tortuosity of the vessels; the margins of the disks were not sharply defined (pseudo neuritis optica). The vision was 20/80 in both eyes; with a + 5.0 cyl., axis 85 it was 20/20 in the right eye and with a + 4.5 cyl., axis 85 it was 20/35 in the left eye. The visual fields were normal. Tension in both eyes was 18 mm. of mercury (Schiotz). Examination of several members of the family did not show any similar findings.

COMMENT

Comparison of the findings in this case with those in the previously cited cases leaves no doubt that I was dealing with the same clinical entity which Duke-Elder⁴ called "rare and peculiar." It is only natural that the mechanism of origin raises considerable speculation. Fleischer⁵ left open the question whether he was dealing with a congenital anomaly, remnants of previous inflammations or deposits of special substances. He doubted whether it would be possible to draw general conclusions about the circulation of the normal cornea. Lutz² was much more positive in evaluating his observations. In spite of the fact that he found identical conditions in a mother and her daughter, making a hereditary anomaly more than probable, he believed the vortex-shaped dystrophy to be caused by inflammatory processes. He referred to Holmes-Spicer,⁵ who drew attention to the fact that the spokelike opacity of the cornea could be formed by edema of the parenchyma. In one of Holmes-Spicer's cases of iritis rheumatica, the opacity extended from the limbus to the center of the cornea, like the spokes of a wheel, but disappeared after three weeks of treatment without leaving any marks. Similar observations were published by Giri,⁶ Blatt⁷ and Hartmann.⁸ All dealt with a spokelike opacity during acute inflammation which cleared up completely after healing of the underlying pathologic process. Lutz, after an extensive discussion of the literature about the fluid metabolism of the cornea, came to the conclusion that in all these cases one is dealing with preformed channels of mechanical filtration which originate at the limbus and flow into the anterior chamber. He assumed that the same system is present in the normal eye, the normal closure of the lid furnishing the motor power for this flow.

Vogels³ came to the conclusion, with which I am inclined to agree, that she was dealing with a special type of a rare corneal dystrophy. The lack of any signs of inflammation, the course of the disease and

4. Duke-Elder, W. S.: *Textbook of Ophthalmology*, St. Louis, C. V. Mosby Company, 1938, vol. 2.

5. Holmes-Spicer, R. L.: *Roy. London Ophth. Hosp. Rep.* **14**:347, 1896.

6. Giri, D. V.: *Tr. Ophth. Soc. U. Kingdom* **39**:145, 1919.

7. Blatt, N.: *Klin. Monatsbl. f. Augenh.* **80**:472, 1928.

8. Hartmann, E.: *Ann. d'ocul.* **161**:161, 1924.

the factor of heredity made such a diagnosis more than probable to her. She considered the peculiar whirlpool-like formation a complete riddle and declined to make any speculative, far reaching conclusions. On the other hand, she tried to clear up the question as to the nature of the pigment. Biopsy of a small piece of the superficial corneal layers led to the conclusion that she was dealing with glycogen, because there were light-diffracting droplets below the epithelium. After staining a section with compound solution of iodine, she found brownish granules. Another section, stained with this solution after extensive watering, did not show any staining.

I do not believe that Vogel's conclusions with regard to the glycogen are convincing. The light-diffracting droplets could have been one of several chemical components, and the granules stained with compound solution of iodine looked to me like the unstained original yellowish brown spots, which were removed mechanically through the intensive watering of the second specimen. Glycogen would be an extremely unusual substance to find in corneal tissue. It is the unutilized storage form of carbohydrates for a metabolic activity which does not exist in the cornea, according to present knowledge. The only place in the eye where glycogen has been reported is the myoid part of the retina, and this was doubted by such an expert in glycogen as Best.⁹ Unusual collections of glycogen appear in a rare metabolic disturbance, which is called Gierke's disease. Apparently none of the patients in the previously reported cases and definitely not my patient was afflicted with the disease. Therefore I came to the conclusion that the yellowish brown spots must be some other substance. I am of the opinion that they are melanin, a brown or black pigment which occurs normally and pathologically in the ocular tissues. Melanin is a near relation to epinephrine, and in 1913 Masson¹⁰ demonstrated the colorless mother substance of melanoblastic cells in the normal conjunctiva. Through silver impregnation they became oxidized into melanin. In 1922 Redslob¹¹ proved the deeper cells of the limbal conjunctiva to be potential menoblasts. They can be converted into melanin through some oxidizing ferments, the so-called dopa reaction. That this process is occasionally reversible can be proved through the following casual observation.

In 1934 a Negro came under my observation for a slight acid burn of his right conjunctiva. The limbus conjunctivae of both eyes was surrounded by a large girdle of pigment. In his right cornea an oval-shaped dense patch of brown pigment stretched for 3 mm. at 11 o'clock toward the center. The pigment was located apparently just above Bowman's membrane. Even though the acute

9. Best, F.: *Centralbl. f. allg. Path. u. path. Anat.* **18**:465, 1907.

10. Masson: *Compt. rend. Soc. de biol.* **75**:210, 1913.

11. Redslob, E.: *Ann. d'ocul.* **159**:523, 1922.

inflammation cleared up after a few weeks and the eyes were found to be normal, I had occasion to reexamine him at certain intervals, for the last time in August 1939. I then found that the brown path had become channeled by clear intervals, beginning at the central part of it and not yet reaching the limbus corneae at any point.

This observation gives a clue to the origin of the whirlpool-like opacity in my case. One may assume that originally a sheet of melanin granules covered Bowman's membrane and that some unknown agent

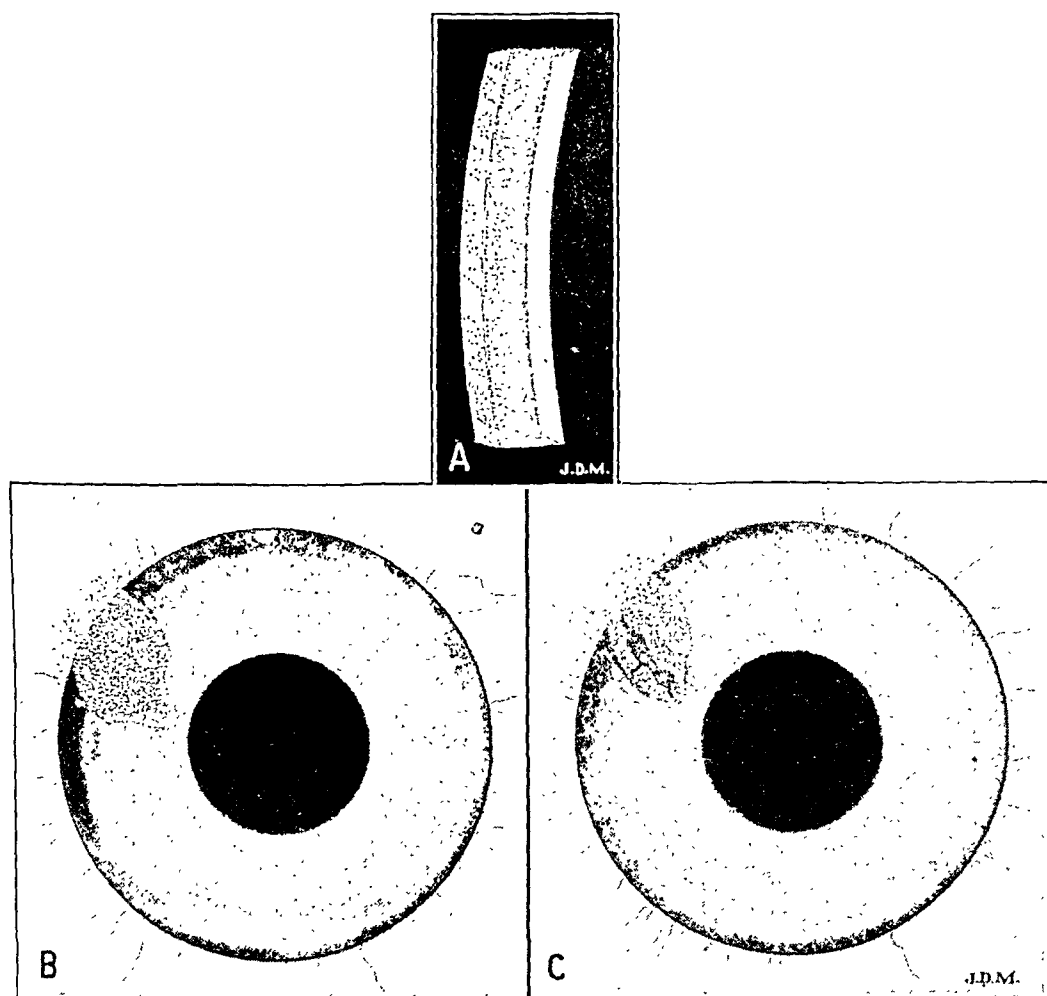


Fig. 2.—*A*, section of the right cornea as seen through the slit lamp. *B*, appearance of the right cornea in 1935. *C*, appearance of the right cornea in 1939.

filtering from the anterior chamber formed the clear channels either by dissolving the melanin or by converting it into its colorless mother substance. The picture of the upper half of the left cornea in my case makes this explanation plausible.

It seems to me difficult to explain the whirlpool-like formation of these channels. Looking for analogous formations in other parts of the human body, I find the crown of human hair and the lines of the

finger tips in similar arrangements. In the skull and in the finger tips, skin covers a dome-shaped structure; in the latter, this structure consists of muscles, and in the former, of bones. I believe that a similar arrangement exists in the cornea, the dome-shaped structure being represented by stroma covered with epithelium. It has been known for a long time that many organs, like the spongiosa of the head of the femur or the aorta, have a definite functional structure, obeying mathematical laws of mechanics. In 1935 Kokott¹² established the fact that such a functional structure exists in the sclera. One may assume that a similar structure exists in the cornea to maintain equal refraction in spite of the continuous fluctuation of the intraocular tension. The pathologic impregnation with pigment made this structure visible. It would be desirable to prove the existence of such a functional structure experimentally and mathematically.

SUMMARY

For the first time in the English-American literature a case of the "rare and peculiar" vortex-shaped corneal dystrophy has been described. As in the 4 previously reported cases, the patient was a woman with hyperopic astigmatism. In my opinion, the colored material is melanin, and the whirlpool-like arrangement is caused by the functional structure of the corneal tissue.

12. Kokott, W.: *Klin. Monatsbl. f. Augenh.* **94**:33, 1935.

POSTSCRIPT ON IMAGE EXPANSION BY THE FOVEAL CLIVUS

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DETROIT

I recently demonstrated the untenability of the usual explanation of the foveal depression,¹ which holds the latter to be simply an excavation of tissue which would interfere with optimal function of the macular center as a region of maximal resolving power. It was suggested that the purpose of the depression, at least where it is deep and convexiculate, as in Sauropsida, is to expand the image locally by a peripheral refraction of the eikonogenic rays where they strike the internal limiting membrane along the clival surfaces.

At the time, I had only logical grounds for believing the refractive index of the retina to be higher than that of the vitreous—a prerequisite for the postulated magnifying action of the foveal pit—and was unaware that actual measurements of retinal indexes had long since been made by Valentin.²

Valentin admonished against ignoring the disparity of the retinal and vitreous refractive indexes in fine calculations and against treating the intraocular course of light rays as if they stopped at the internal limiting membrane. He did not suggest any possible effects of the disparity, however.

Matthiessen³ knew of Valentin's determinations (though he sadly misquotes them) and stated that while he was able to confirm them they "have no significance for dioptrics." It was perhaps Matthiessen's authority which kept the data from being perpetuated into modern textbooks.

Valentin measured the refractive indexes of the retinas of a number of vertebrates, including 6 mammals, for which the values ranged from 1.3407 for the green monkey to 1.3460 for the dog, the average for the

From the Ophthalmic Research Laboratory, Wayne University College of Medicine.

1. Walls, G. L.: Significance of the Foveal Depression, *Arch. Ophth.* **18**: 912 (Dec.) 1937.

2. Valentin, G.: Ein Beitrag zur Kenntnis der Brechungsverhältnisse der Thiergewebe, *Arch. f. d. ges. Physiol.* **19**:78, 1879; *Fortgesetzte Untersuchungen über die Brechungsverhältnisse der Thiergewebe*, *ibid.* **20**:283, 1879.

3. Matthiessen, L.: Die neueren Fortschritte in unserer Kenntnis von den optischen Baue des Auges der Wirbeltiere, in *Festschrift zur Feier des siebenzigsten Geburtstages von Hermann von Helmholtz*, Leipzig, L. Voss, 1891, p. 51.

mammals being 1.34385. The values for 4 and 7 month human fetuses averaged 1.3486; but it is probable that the value for the adult human being is lower, since during development in the chick the index sinks from 1.3476 (average for 12, 14 and 16 day embryos) to 1.3461 for the young adult. The average for the 2 primates studied, the green monkey and the baboon, was 1.34265, and the index for the adult human being may prove to be close to this.

Rather higher values are found for animals other than mammals. The maximum found by Valentin was 1.3794 for a frog (*Rana esculenta*), freshly caught in May. A specimen caught in October gave a much lower figure, 1.3416. The diet, especially its fat content, may have a considerable influence, and experiments to test this possibility would be most interesting.

The important figures for consideration here are those for the only foveate bird examined by Valentin, a species of parrot (*Chrysotus aestiva*). The retina of this form has an index of 1.3475; the vitreous, an index of 1.3391. By substituting these values in the computations based on figure 6 in the exposition of my theory,¹ angle *BAC* proves to be 1.64 degrees, which would afford an areal expansion of the image in the middle of the hawk's fovea of approximately 30 per cent.

The attendant local increase in resolving power appears adequate to make the magnification of the image by the clival surface of the foveal pit seem a sufficient teleologic reason for the existence of the pit, at least when it is shaped as it is in most birds and lizards.

In the concaviclivate human fovea, however, the much less precipitous clival slope and the presumptive lower refractive index would appear to make for a degree of image expansion too low to account for the evolution of the human foveal pit from no depression at all *directly* to its present gentle profile. Wherever the foveas of animals approach the fovea of man in shallowness, they can invariably be shown to have been deeper in the ancestors. In the light of the resurrected data of Valentin, it seems more likely than ever that the human fovea has degenerated, like that of *Sphenodon*, the owls and the pigeon,¹ from a once much more deep and abrupt depression. The possibility that the higher apes have less gradual foveas than man seems fairly strong, and it is to be hoped that studies of them may soon be made.

Many a primate has a more "diurnal" eye than man, with higher cone to rod ratios; and since retinal adaptations for diurnality and for visual acuity go hand in hand, it should not be surprising if some other primates prove to have better foveas than even the phenomenally sharp-sighted Hottentot.

OPERATION ON THE OCULAR MUSCLES

A FEW POINTS IN OPERATIVE TECHNIC

MOSES FREIBERGER, M.D.

NEW YORK

Simple modifications of the technic of operations on the ocular muscles, I have found, can materially improve the results obtained and reduce the time required. I report these for what they may be worth to others. The points made may appear simple and obvious, but they represent thought and experience, and a trial will prove their value. I am aware of the many operations on the ocular muscles, and I have found procedures of merit in many of them.

When Jameson¹ introduced his epoch-making recession operation, supplementing simple tenotomy, there grew up a literature on various modifications of this technic. Each writer described what he regarded as a simpler method of executing the essential principle of reattaching the cut muscle at a given point on the globe. These modifications are welcome additions, for in each case one may find points of value.

In 1933 Curdy² introduced a simplified technic for muscle recession. He advised a horizontal conjunctival incision instead of the vertical one and attachment of the muscle to the sclera with a single suture. I had anticipated Curdy's work and in the fall of 1932 had presented a similar operation at a monthly staff meeting at the New York Ophthalmic Hospital.

The technic of this recession operation as I have performed it is as follows: The conjunctival incision is made horizontally, extending from near the limbus at the 180 degree axis back to the plica semilunaris and somewhat beneath it. One of the advantages of a horizontal incision over a vertical one is that less bleeding is encountered owing to the fact that the incision runs parallel to the conjunctival vessels and that one can often avoid the vessels by cutting between them. Another advantage is the inconspicuous scar resulting. I find that frequently after healing it is almost impossible to detect the site of the incision. In making the incision, the legs of the open forceps should be placed in the direction in which the line of incision is to be made (fig. 1), so that in closing them a vertical fold of conjunctiva is grasped and readily nicked in the horizontal direction desired.

1. Jameson, P. C.: Arch. Ophth. **51**:421 (Sept.) 1922.

2. Curdy, R. J.: Am. J. Ophth. **16**:890 (Oct.) 1933.

The conjunctiva is now undermined well back over the muscle and toward the cornea. Tenon's capsule is buttonholed, and the muscle forceps is slipped under the tendon immediately behind the insertion in the usual manner. The muscle forceps is placed in position to grasp the well spread muscle. To facilitate this procedure, it is well to hold the female blade of the forceps alone and pass it beneath the muscle. This is as simple as inserting a muscle hook. After the tendon is cut, it is wise to pass the muscle hook all around the area of the insertion to make sure that no strands of muscle are still attached to the sclera. Any such strands left intact might cause failure of the operation.

A single-armed suture of no. 1 silk or 000 catgut enters the conjunctival wound from above and close to the plica. The assistant holds the conjunctiva aside while the surgeon grasps the muscle forceps and

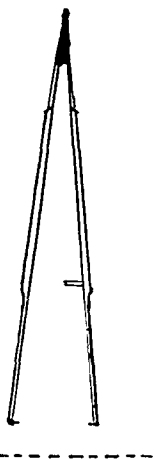


Fig. 1.—The open forceps in relation to the line of incision.

passes the suture through the muscle from above. The suture should be so placed that it will hold a bit more than its middle third. The surgeon then allows the assistant to hold the muscle forceps while he grasps the stump with the fixation forceps to steady the globe.

The distance back on the sclera that the muscle must be receded is now noted. At this point the needle enters the superficial layer of the sclera in a vertical direction. The fixation forceps is then removed from the stump, the muscle forceps is held by the surgeon and the suture is continued through the muscle from its undersurface outward and on through the conjunctival wound opposite the initial puncture. When this suture is tied, not too tightly, it holds the muscle in place at its new insertion. It also closes the posterior end of the conjunctival wound. One or two additional sutures are usually required to close the wound completely (fig. 2).

Some doubt was raised when I first introduced this operation as to whether a single scleral bite is sufficient to hold the muscle to the sclera and whether the muscle would be spread sufficiently without additional sutures. I tried to satisfy myself and others on these points when I had occasion to do this operation on an old blind eye that was enucleated two weeks later. My patient consented to have me enucleate his eye in "two stages." At the time of the enucleation I allowed a goodly portion of the receded muscle to come away with the globe. I was able to demonstrate how well spread the muscle was at its attachment to the globe and, above

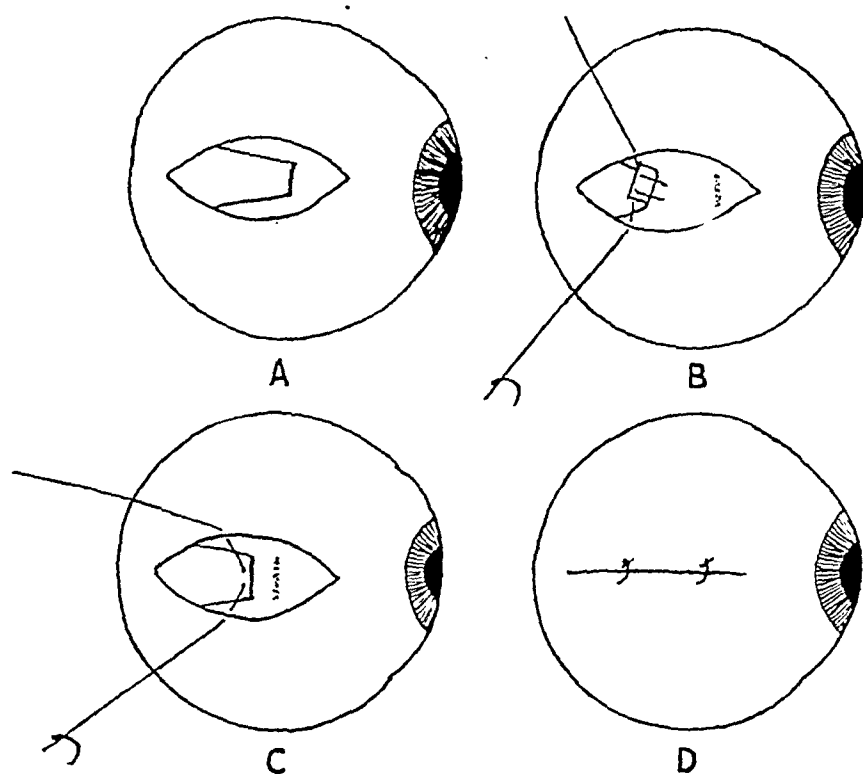


Fig. 2.—Diagrammatic representation of operative technic. In *A*, a horizontal incision (oval conjunctival wound) is made, exposing the sclera and the muscle. In *B*, the muscle is cut away from its insertion. The suture is placed in the conjunctiva, muscle and sclera, back to the muscle and out through the conjunctiva. In *C*, the muscle is resting on the sclera at its new attachment, with a single suture holding it firmly in place. In *D*, the suture is tied, uniting the margins of the conjunctiva and closing the wound. An additional suture is usually required to close the wound completely.

all, how firmly adherent it was. It required considerable power to pull it away from the sclera.

An outstanding feature of the operation is that it necessitates only one suture through the sclera. This is a distinct advantage. The entire operation is easily performed, and the time consumed is reduced to a minimum. Since 1932 my associates and I have performed over 300

recessions by this method. In view of the ease of execution, the minimum consumption of time and the good results, it must be acknowledged that this operation has merit.

With reference to the suture material used at operations on muscles, I make it a rule to use catgut in children under 8 years of age and silk in older patients. The sutures themselves are seldom longer than 6 inches (15 cm.) and at this length are easily handled and their exposure to contamination minimized. With reference to tying the sutures with

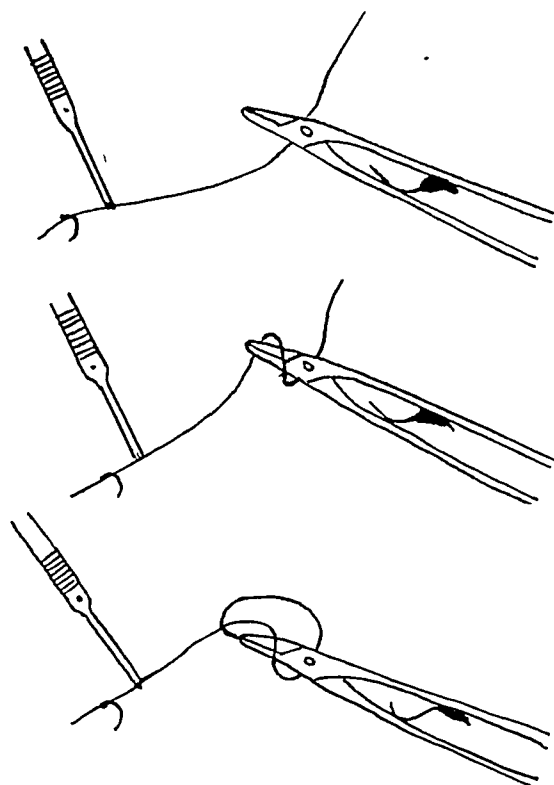


Fig. 3.—Steps in tying sutures with instruments.

instruments, I feel that this simple procedure should hardly be called to the attention of the ocular surgeon, but from frequent observation I have found that it is often done incorrectly. Figure 3 shows in a diagrammatic manner the steps, beginning with the placing of the needle holder between the short and the long end of the suture and not from the outer side of the long end. The short end need not be more than 1 or $1\frac{1}{2}$ inches (2.5 or 3.8 cm.) from the wound. The long end should be grasped by the tissue forceps and wound once around the head of the needle holder; then the needle holder grasps the end of the short arm, pulls it through the loop and completes the knot. The second tie is carried out in the same manner.

For the resection operation, the method of Reese has been a standard procedure and is still popular. This operation, however, has some shortcomings which pave the way for improved technic. For example, the central double-armed suture, in passing over the muscle end and under the stump, must necessarily lie between the edge of the muscle and the stump when these structures are brought end to end. The interposition of the suture does not allow a close approximation of the muscle and stump. After all sutures are tied, there is a boggy mass of tissue due to the inclusion of excess conjunctiva, muscle and stump in the suture. The end result is a heavy, prominent scar.

I have modified the technic in my work. I use the same suture material as in the Reese operation. For the central double-armed suture, I use no. 3 braided silk, and for the lateral sutures, no. 1 braided silk. The conjunctival incision is made in the usual vertical direction and the posterior flap undermined. It is here that care must be exercised not to dissect the posterior flap too extensively, for this would have the effect

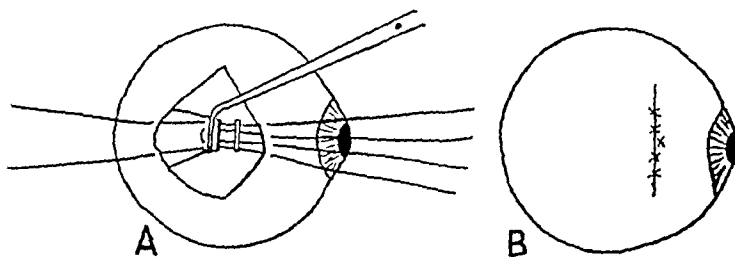


Fig. 4.—*A*, diagrammatic drawing showing the position of the sutures at the resection operation. *B*, location of the sutures after closure of the wound.

of a sliding flap, which would cause the massing of an excess conjunctiva and overlapping of the edges when the wound is closed. The flap should be dissected only over the area of the muscle and down its edges, so that the muscle alone is freed from its adjacent tissues. The anterior flap needs no undermining.

The muscle thus isolated is held on a muscle forceps while the desired portion is resected. The central double-armed suture is inserted in its middle third. The suture is introduced from above and does not include the conjunctiva. It is then continued through the middle third of the stump, on through the anterior conjunctival flap close to its margin, there to be tied later. The marginal sutures are passed in the same manner as in the Reese operation and include the conjunctival margin of the posterior flap, but the sutures should not be separated more than the width of the muscle (fig. 4 *A*).

While the assistant holds both marginal sutures, which tends to pull the eyeball forward, the surgeon ties the middle suture by hand, keeping the index fingers close to the knot so as to minimize the danger of tear-

ing the suture. The tying of this suture draws the muscle and stump together and approximates their edges. The marginal sutures are now tied. They reenforce the central suture and at the same time approximate the adjacent conjunctival margins. Two additional sutures of no. 1 silk may be required, one on either side, to close the wound completely (fig. 4 *B*). Any small areas of gaping conjunctiva between these sutures can be cemented by pinching them with the tissue forceps.

The modified technics described herein have been used by my associates and by myself on many occasions and have invariably given such satisfaction that I felt they had sufficient merit to warrant their description.

Clinical Notes

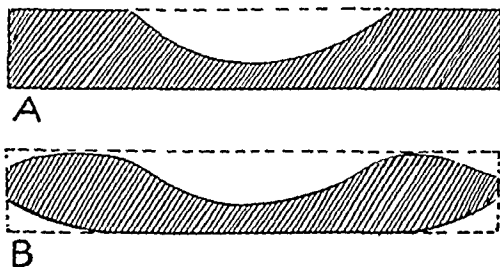
A NEW LENS

ANDREW HUNTER, M.D., MCKEESPORT, PA.

A new lens is here presented which will overcome the objections to the appearance of strong concave lenses and especially do away with the sharp line which marks the periphery of the concave part of the myodisk lens.

This lens is similar to the myodisk lens, but the sharp edge of the concave part of the latter, which is so conspicuous, is replaced by a gradual curve. The margin of the lens is also cut down by a gradual curve to the thickness which is suitable for rimless mountings.

Possibly originality may not be claimed for cutting down the periphery of these concave lenses, as others may have done some work



A, cross section of the myodisk lens. *B*, cross section of the new lens.

in that direction. The distinctive feature of the lens is that the sharp edge of the myodisk lens is cut down and is replaced with a gradual curve which is not so conspicuous. The accompanying diagram shows cross sections of both lenses.

Representatives of two leading optical houses have stated that such a lens is not practicable, but patients seeking cosmetic improvement seem to like it.

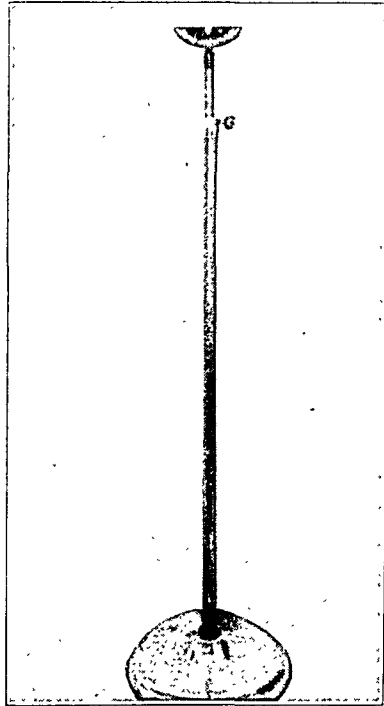
These lenses have been made for me by the B. K. Elliott Company, of Pittsburgh. They are largely handmade, and the average wholesale price is about \$10 a pair.

A SIMPLE AND INEXPENSIVE CHIN REST FOR USE WITH A TANGENT SCREEN

LOUIS C. RAVIN, M.D., TOLEDO, OHIO

In taking central fields with a Bjerrum screen it has seemed desirable to use a chin rest. This not only makes the patient more comfortable but insures better fixation of the central fixation target.

The use of an ophthalmic instrument table with a clamped-on chin rest, such as can be purchased from a manufacturer of optical instruments, has proved to be satisfactory. It was my desire, however, to make a less bulky and less expensive chin rest for use in the office. Practically any floor lamp stand with a thumbscrew adjustment, as illustrated here, may be used. The flexible tubing with the lighting fixture is removed. The movable tubular shaft may be replaced with an iron bar, as I have done, or it may be used as it is. The chin rest piece¹ is then attached to the rod shaft by grinding the shaft down at one end to fit into the opening in the chin rest. The small screw in



Chin rest for use with the tangent screen.

the chin rest piece is tightened, thus fixing it to the rod. If a solid iron rod is not easily obtained, a small piece of solid metal rod may be fitted into the tubular shaft and held in position with a small bolt or rivet. The end of this solid bar is then ground to fit into the chin rest piece. If one does not have the inclination or the facilities to do this bit of metal handiwork, any metal workshop can do this at a reasonable price.

The stand fits easily into a small space, is simple to use and is inexpensive.

316 Michigan Street.

1. Purchased from Bausch & Lomb Optical Co.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

SOME BASIC PRINCIPLES OF DARK ADAPTATION

ROBB McDONALD, M.D.*

PHILADELPHIA

The physiology of rod and cone vision has been under investigation for many years, and many significant contributions have been made within the past few years, notably by Hecht and his associates in this country and Lythgoe and his associates in Great Britain. These studies, combined with the increasing knowledge of the biophysics of the eye and the chemistry of the vitamins, enable one to approach the problem of dark adaptation and its clinical significance without the skepticism manifest a few years ago. The greater part of this investigative work has been published in journals not always accessible to ophthalmologists, and frequently an advanced knowledge of physics and chemistry is necessary to interpret the findings. In this paper an attempt is made to review the problem in a brief and simplified manner and to give the present status of dark adaptation and the chemical basis of vision.

Schultze¹ in 1866 was the first to suggest that the retina possesses two types of receptor cells, but it was not until 1895 that the duplicity theory was finally established by von Kries.² In brief, it proposed that the cones are concerned chiefly with visual acuity and color discrimination at high intensities of illumination, while the rods are responsible for vision at low intensities of illumination, at which time the vision is most effective in the periphery of the retina and is colorless. Parsons introduced the terms photopic for vision at high intensities of illumination and scotopic for vision at low intensities of illumination. Therefore, photopic vision is essentially cone vision and is chromatic, and scotopic is essentially rod vision and is achromatic.

* Research Fellow in Ophthalmology.

From the Department of Ophthalmology, the University of Pennsylvania.

This work was supported by a grant from the John and Mary Markle Foundation.

1. Schultze, M.: Zur Anatomie und Physiologie der Retina, Arch. f. mikr. Anat. 2:175, 1866.

2. von Kries, J.: Ueber die Funktion der Netzhautstäbchen, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. 9:81, 1895.

The effect of abrupt changes from light to darkness is to lower the visual acuity; this phenomenon is well known. Coincident with the lowering of visual acuity, there is an increase in the light sensibility of the peripheral portion of the retina. This quantitative increase in the retinal sensitivity is known as dark adaptation. Dark adaptation is measured by determining the minimum intensity of a small area of light which is just perceptible to the eye at different time intervals after exclusion of all other light. This minimum intensity which is just perceptible is called the threshold. Hecht has emphasized that the factors to be considered in any determination of dark adaptation are: (1) the intensity and duration of the preadapting light and (2) the area and wavelength of the testing light, the duration of its exposure, the region of the retina stimulated, the size of the pupil and the exact intensity of the test light at any time during the course of dark adaptation.

Aubert³ in 1865 was the first to describe the increased sensitivity of the eye when it is kept in the dark. During the latter part of the nineteenth century other investigators, notably König and Ritter⁴ and Abney and Festing,⁵ were concerned chiefly with the wavelength and the threshold of light just necessary to be visible in the dark-adapted eye. This work has been repeated several times since then, the most accurate determinations being those of Hecht and Williams⁶ and of Kohlrausch.⁷ They took into consideration the energy distribution of the spectrum (which the earlier workers did also, but they were handicapped by lack of modern apparatus) and found that the maximum visibility at low intensities was reached with light of a wavelength of 510 millimicrons. The same procedure has been used to determine the maximum visibility at high intensities, but here it was found by measuring the relative energy at different wavelengths required to match a high constant brightness. The maximum visibility was shown to be at 555 millimicrons, which is nearer the red end of the spectrum; this shift is the well

3. Aubert, H.: *Physiologie der Netzhaut*, Breslau, E. Morgaster, 1865.

4. König, A., and Ritter, R.: *Ueber den Helligkeitswerthe der Spektralfarben bei verschiedener absoluter Intensität*, in *Festschrift zur Feier des siebenzigsten-Geburtstages von Hermann von Helmholtz*, Leipzig, L. Voss, 1891, p. 309.

5. Abney, W. de W., and Festing, E. R.: *Color Photometry*, Proc. Roy. Soc. London **50**:371, 1891.

6. Hecht, S., and Williams, R. E.: *The Visibility of Monochromatic Radiation and the Absorption Spectrum of Visual Purple*, J. Gen. Physiol. **5**:1 (Sept.) 1922.

7. Kohlrausch, A.: *Untersuchungen mit farbigen Schwellenprüflichtern über den Dunkeladaptationsverlauf des normalen Auges*, Arch. f. d. ges. Physiol. **196**: 113. 1922.

known Purkinje phenomenon. Chart 1 shows the relation between the wavelength and the relative energy required to produce a specific brightness at high and at low intensities.

This shows the well known phenomenon that the light-adapted and dark-adapted eyes have nearly the same threshold for red light. In other words, the color-colorless or photochromatic interval is longest for the short waves, i. e., violet and blue, while the interval for red is very small, i. e., "red crosses the threshold colored." The importance of this phenomenon is apparent. If the test flashes are made with a red light of 680 millimicrons, the results will record practically cone

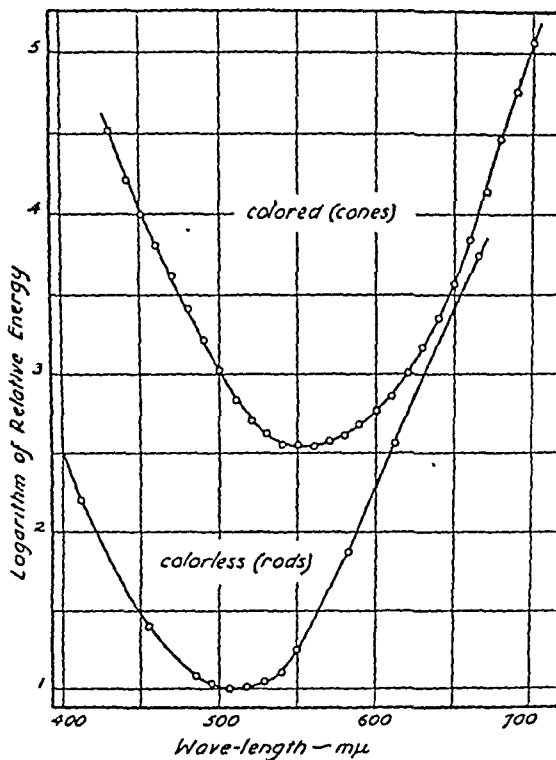


Chart 1.—Relation between the wavelength and the relative energy required to produce a specific visual effect at high and low illuminations. (From Hecht, S.: Rods, Cones, and the Chemical Basis of Vision, *Physiol. Rev.* **17**:242, 1937.)

behavior only. When blue light is used, one will obtain the maximum function of the rods (chart 2). If one reduces the intensity of a light by reducing the amount of current, the color of the light will change, especially at an extremely low intensity. Hence it is important that the intensity of the test light be reduced by filters of a neutral tint rather than by reducing the current by means of a rheostat.

The earlier workers did not recognize the factor of cone adaptation, the principal reason being that it takes place rapidly, and they plotted their results on a linear scale as sensitivity of the retina, which method

of plotting obscured their findings. Hecht has recalculated Piper's⁸ data, which show, along with the work done by him and his co-workers,⁹ that the cones do adapt themselves. Hecht's measurements of cone adaptation were made with red light and also with small 2 degree fields centrally located, both of which show the character and the course of cone adaptation.

The importance of adequate fixation and of knowing what portion of the retina is being stimulated by the test flash is shown in chart 3. With the eye completely dark adapted, the threshold for a 2 degree retinal field was determined at the fovea and at various points in the nasal and temporal portions of the retina. This shows clearly that the more peripheral the test flash the lower the threshold, owing to the increasing predominance of rods in the peripheral portion of the retina.

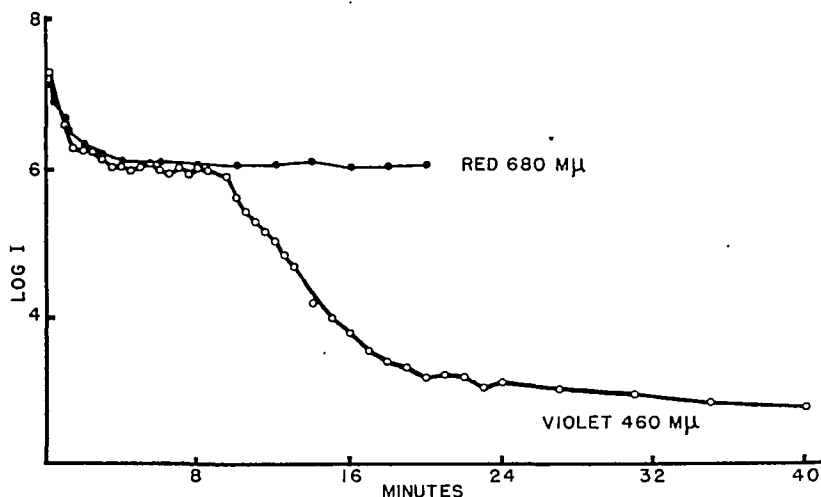


Chart 2.—Course of dark adaptation following light adaptation to 4,900 millilamberts. The solid circles represent the course as measured with red light of 680 millimicrons; the open circles, as measured with violet light of 460 millimicrons.

The control of the retinal location of the test flash by adequate fixation is one important feature which is absent in most of the popular instruments now used in clinical work. Sloan¹⁰ recently reviewed the technics

8. Piper, H.: Ueber Dunkeladaptation, *Ztschr. f. Psychol. u. Physiol. d. Sinnesorg.* **31**:161, 1903.

9. Hecht, S.: The Nature of Foveal Dark Adaptation, *J. Gen. Physiol.* **4**:113 (Nov.) 1921. Hecht, S.; Haig, C., and Chase, A. M.: The Influence of Light Adaptation on Subsequent Dark Adaptation of the Eye, *ibid.* **20**:831 (July) 1937. Hecht, S.; Haig, C., and Wald, G.: Dark Adaptation of Retinal Fields of Different Size and Location, *ibid.* **19**:321 (Nov.) 1935.

10. Sloan, L. L.: Instruments and Technique for the Clinical Testing of Light Sense: III. Control Fixation in the Dark-Adapted Eye, *Arch. Ophth.* **22**:228 (Aug.) 1939.

for the clinical testing of the light sense and emphasized the importance of maintaining fixation. The subjective difficulties of this alone are apparent.

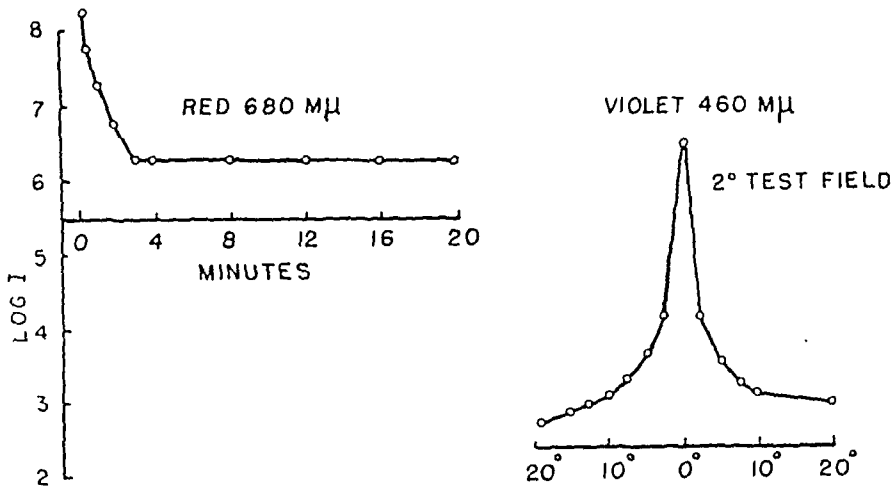


Chart 3.—The left part of the chart represents the course of dark adaptation as measured with red light. With the eye completely dark adapted and using a violet filter in the test beam, the threshold (right side of the chart) was determined at the fovea and at various points up to 20 degrees on either side of the fovea.

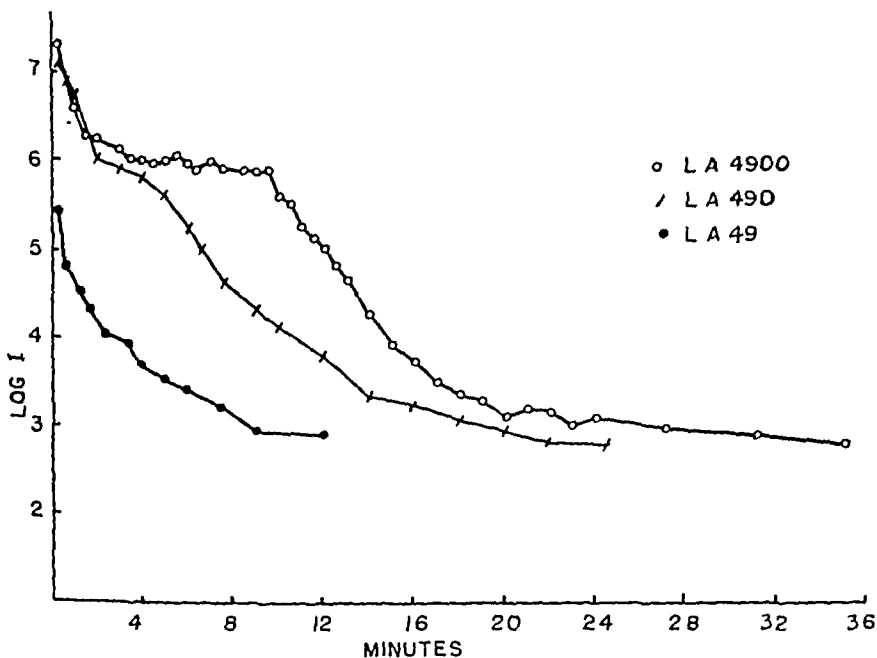


Chart 4.—Course of dark adaptation following various intensities of light adaptation. (The numbers in the right hand corner represent the intensity in millilamberts.)

Dark adaptation presupposes light adaptation. By increasing the intensity or time of the preadapting light, the recovery is naturally delayed. Chart 4 shows this clearly. The higher the intensity of the

preadapting light, the higher the initial threshold and the more prominent the cone adaptation. The rod-cone transition time is also delayed. The initial rod adaptation is masked in the cone adaptation.

Chart 5 shows the course of dark adaptation in a normal person as demonstrated with the Hecht apparatus. Haig, Hecht and Patek¹¹ expressed the belief that it is important to be able to show both rod and cone adaptation adequately in the same curve. The first part of the curve represents the adaptation of the cones, and the plateau is known as the cone plateau. The time of the sudden break in the curve is known as the rod-cone transition time, while the lower curve represents the adaptation of the rods and the lower plateau is called the final rod threshold. It is this threshold that most of the popular instruments

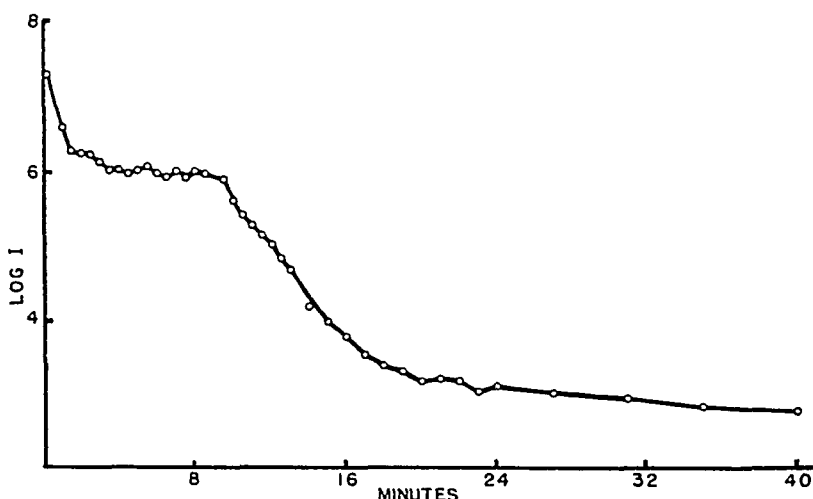


Chart 5.—Course of dark adaptation in a normal person following light adaptation at 4,900 millilamberts. The test flash subtended a retinal field 3 degrees in diameter and 7.5 degrees to the nasal side of the fovea. The test was made with a violet filter in the beam, and the test light was exposed for one-fifth second.

are concerned in measuring and that is the most reproducible in the clinical determination of dark adaptation, though its value is dubious unless the retinal location of the test flash is adequately maintained.

The remaining factors that must be considered are the area and duration of exposure of the testing light and the area of the pupil. For short exposures and small areas, the product of the intensity and the exposure time are constant (Graham and Margaria¹²). In most

11. Haig, C.; Hecht, S., and Patek, A. J.: Vitamin A and Rod-Cone Adaptation in Cirrhosis of the Liver, *Science* **87**:534 (June 10) 1938.

12. Graham, C. H., and Margaria, R.: Area and the Intensity-Time Relation in the Peripheral Retina, *Am. J. Physiol.* **113**:299 (Oct.) 1935.

instruments now in use an area exactly large enough to give a uniform cross section of the retina is exposed just long enough to be within the physiologic limits of the retinal action time. The size of the pupil is usually taken into account for accurate investigations, as the physiologic properties of the retina are concerned with apparent brightness rather than external brightness. The apparent brightness is expressed as external brightness in millilamberts times $10/\pi$ times pupil area in square millimeters, the unit being a photon. The use of an artificial pupil, the fixation of the pupil with miotics and photographing the pupil and computing its area have all been utilized. However, the pupil changes in size so rapidly from light to dark adaptation that except in precise work it need not be taken into account, though if the pupil is miotic and fixed, as in Argyll Robertson pupils, the threshold will be generally raised as the retinal illumination is reduced.

Thus any instrument for the clinical determination of dark adaptation should take into account the intensity and duration of the pre-adapting light and the area, color, retinal location and duration of the testing light. These can all be made constant, the only variable being an adequate range of intensity of the test light. The intensity should be plotted on a logarithmic scale against time in minutes as the abscissa. The use of a logarithmic plot is necessary because of the tremendous increase in sensitivity of the retina, an increase which may be on the order of 100,000/1. Plotted on a linear scale, the true course of dark adaptation is masked.

With the discovery of visual purple by Boll¹³ in 1876 and its extraction from the retina by Kuehne¹⁴ in 1879, it was natural to associate it with the visual process. In fact, Parinaud¹⁵ in 1881 associated night blindness with deficiency in visual purple. The earlier workers, however, were chiefly concerned with the absorption spectrum of visual purple. If visual purple is the photosensitive substance responsible for rod vision, then the primary law of photochemistry should be fulfilled; that is, that only that light which is absorbed can initiate a chemical change. The absorption spectrum of visual purple and the spectral sensitivity of the eye at low intensities are closely related—a fact which one would expect to find if visual purple is responsible for the chemical initiation of the visual response. This relation is demonstrated in

13. Boll, F.: *Zur Anatomie und Physiologie der Retina*, Monatsber. d. berl. Akad., Nov. 12, 1876, p. 783.

14. Kuehne, W.: *Chemische Vorgänge in der Netzhaut*, in Hermann, L.: *Handbuch der Physiologie*, Leipzig, F. C. W. Vogel, 1879, vol. 3, p. 235.

15. Parinaud, H.: *L'héméralopie et les fonctions du pourpre visuel*, Compt. rend. Acad. d. sc. 93:286, 1881.

chart 2 of an article by Ludvigh.¹⁶ The minimum energy required to produce a bleaching effect on visual purple has also been determined and closely follows the curve for the absorption spectrum. Thus the close correlation between the minimum energy required to produce the sensation of light, the absorption spectrum and rate of bleaching of visual purple all point to it being the photosensitive substance in the rods responsible for the increased sensitivity in dark adaptation. Wald¹⁷ recently isolated a photosensitive substance in the cones which he has called iodopsin and which is chemically distinct from visual purple.

Kuehne, in his classic monograph, described the bleaching of visual purple when acted on by light and its recovery when kept in the dark. Practically all his original observations have been confirmed, and a great deal has been added to the knowledge of the chemistry of visual purple within the past few years. It is generally accepted that visual purple is a conjugated carotenoid protein; its molecular weight is about 800,000 as determined by the diffusion method (Hecht, Chase and Shlaer¹⁸), though Lythgoe and Goodeve¹⁹ found it to be nearer 100,000 with the ultra centrifuge. It has also been determined that visual purple may regenerate in two ways, directly from its photoproducts or from newly supplied material. The discovery by Fridericia and Holm²⁰ and of Tansley²¹ that the rate of regeneration of visual purple in animals on a vitamin A-deficient diet was reduced and the experiments of Yudkin, Kriss and Smith²² in relieving vitamin A deficiency by feeding whole retina proved rather conclusively that vitamin A must in some way be concerned with the visual response. Confirmation of this was definitely established by Wald²³ when he isolated vitamin A from the

16. Ludvigh, E.: Determination and Significance of the Scotopic Retinal Visibility Curve, *Arch. Ophth.* **20**:713 (Nov.) 1938.

17. Wald, G.: Photo-Habile Pigments of the Chicken Retina, *Nature*, London **140**:545 (Sept. 25) 1937.

18. Hecht, S.; Chase, A. M., and Shlaer, S.: The Diffusion Coefficient and Molecular Size of Visual Purple, *Science* **85**:567 (June 11) 1937.

19. Lythgoe, R. J., and Goodeve, C. F.: Visual Purple, *Tr. Ophth. Soc. U. Kingdom* **57**:88, 1937.

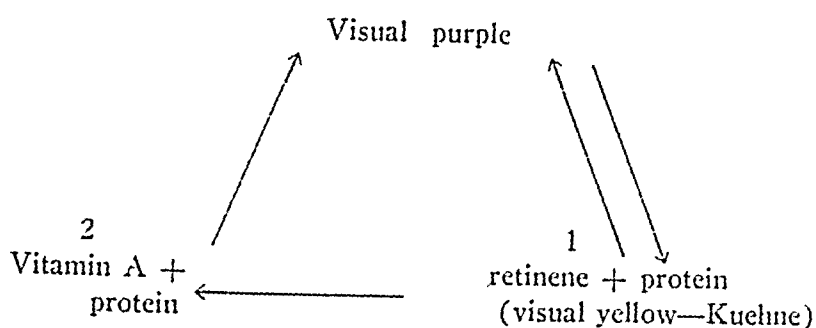
20. Fridericia, L. S., and Holm, E.: Experimental Contribution to the Study of the Relation Between Night Blindness and Malnutrition: Influence of Deficiency of Fat-Soluble A Vitamin in the Diet on the Visual Purple in the Eyes of Rats, *Am. J. Physiol.* **73**:63 (June) 1925.

21. Tansley, K.: The Regeneration of Visual Purple: Its Relation to Dark Adaptation and Night Blindness, *J. Physiol.* **71**:442 (April) 1931.

22. Yudkin, A. M.; Kriss, M., and Smith, A. H.: Vitamin A Potency of Retinal Tissue, *Am. J. Physiol.* **97**:611 (July) 1931.

23. Wald, G.: Vitamin A in Eye Tissues, *J. Gen. Physiol.* **18**:905 (July) 1935; On the Distribution of Vitamins A₁ and A₂, *ibid.* **22**:391 (Jan.) 1939.

retina itself. Wald has suggested the following scheme to illustrate the chemical basis of the visual response:



When light acts on visual purple in the isolated retina or in solution, it is bleached by a photochemical process to visual, or indicator, yellow; this yellow product finally fades out entirely if the light continues, the retinene being converted into vitamin A plus protein, which product was originally called visual white. If the retina at 0 C. is exposed to light, visual yellow is formed but will not be decomposed to its colorless product, nor will it regenerate to form visual purple. However, at 25 C. visual purple can regenerate from retinene, and retinene can fade out entirely to its colorless product. This proves that the initial bleaching of visual purple to retinene is photochemical but that its recovery from retinene or the complete fading of retinene is a thermal process. Vitamin A is found only in small quantities in completely dark-adapted and bleached retinas; its greatest concentration is found in the colorless decomposition product of retinene, which one would expect from the schematic cycle proposed by Wald. The expression of the visual cycle in the form of this equation concedes, of course, that in constant light adaptation a steady state is reached in which the resynthesis of visual purple is of the same proportion as its bleaching by light.

The regeneration of visual purple in solution from its colorless product, as well as its delayed regeneration in cases of vitamin A deficiency, is the most conclusive proof that vitamin A is a precursor of visual purple as well as a product of decomposition. Wald and Clark²⁴ have shown in subjective experiments that the rate of regeneration of visual purple depends on the intensity and time of the preadapting light. If the preadapting light is of low intensity and acts for a short time, the recovery of visual purple is limited to reaction 1 in the visual cycle and is rapid; however, if the preadapting light is of fairly high intensity and acts for a longer period, the recovery follows reaction 2; that is, the recovery of visual purple is more rapid from retinene than from vitamin A + protein.

24. Wald, G., and Clark, A. B.: Visual Adaptation and Chemistry of the Rods, *J. Gen. Physiol.* **21**:93 (Sept.) 1937.

Within the past few years the clinical application of dark adaptation as a test for vitamin A deficiency has been widely accepted. Bitot²⁵ in 1863 was probably the first to recognize that xerosis and the conjunctival lesions which bear his name were often related to night blindness. The relief of night blindness by the ingestion of fish livers has been known since the time of the Egyptians. Hecht and Mandelbaum²⁶ and Wald, Jeghers and Arminio²⁷ in well controlled experiments were able to show conclusively that experimental vitamin A deficiency caused a rise in the visual threshold during dark adaptation. Chart 6 shows the characteristic change in the dark adaptation curve in vitamin A deficiency. The fact that the cone as well as the rod threshold is raised would imply that vitamin A is also concerned in the visual cycle of the photopigments of the cone. Haig, Hecht and Patek¹¹ have

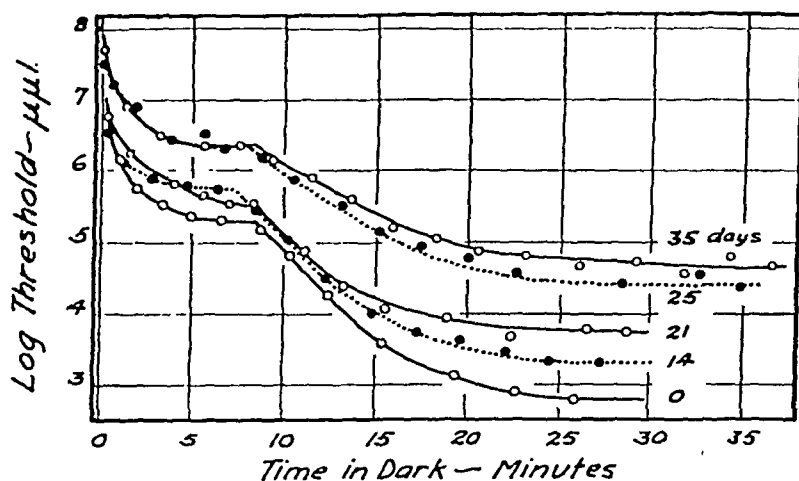


Fig. 6.—Change in the course of human dark adaptation in experimental vitamin A deficiency. (From Hecht, S., and Mandelbaum, J.: *The Relation between Vitamin A and Dark Adaptation*, J. A. M. A. **112**:1910 [May 13] 1939.)

also shown that in cirrhosis of the liver the rod-cone transition time is delayed, a fact which differentiates it from experimentally induced vitamin A deficiency and serves to emphasize that it may be important to obtain the entire adaptation curve with well defined cone adaptation.

The literature abounds with reports of dark adaptation as a test of vitamin A deficiency. Many suggestions have been devised to

25. Bitot: *Mémoire sur une lésion conjunctivale non encore décrite, coïncidant avec l'héméralopie*, Gaz. hebd. de méd. et de chir. **10**:284 (May) 1863.

26. Hecht, S., and Mandelbaum, J.: (a) Rod-Cone Dark Adaptation and Vitamin A, *Science* **88**:219 (Sept. 2) 1938; (b) *The Relation Between Vitamin A and Dark Adaptation*, J. A. M. A. **112**:1910 (May 13) 1939.

27. Wald, G.; Jeghers, H., and Arminio, J.: *An Experiment in Human Dietary Night-Blindness*, Am. J. Physiol. **123**:732 (Sept.) 1938.

simplify and hasten the procedure. It must be remembered, however, that dark adaptation is a physiologic phenomenon that requires time and that there are probably other factors concerned with dark adaptation besides the regeneration of visual purple. Apart from the subjective difficulties of the test, the exact metabolism of vitamin A in the body or the individual daily requirements are not yet known for certain.

Theories do not always fit the facts, and the value of dark adaptation as a routine test of vitamin A deficiency can be established only by a critical analysis of the results obtained. Unfortunately, in one's enthusiasm the basic physiology is sometimes neglected, and a true appreciation of the significant changes is missed.

News and Notes

GENERAL

The following item is taken from a recent number of the *British Medical Journal*:

Prevention of Blindness.—"A great piece of work in the spread of the knowledge of the causes of blindness and the means of preventing this catastrophe was done by the former Prevention of Blindness Committee of the Union of Counties Association of the Blind. When that committee was dissolved the work was taken over by a special committee of the Ministry of Health, which is continuing the analysis of the certificates of blindness prepared for the local authorities throughout the country. There has been some feeling that a voluntary committee, as apart from a Government committee, might serve a useful purpose in driving home the lessons of these statistical returns to masters of industry and local authorities. Accordingly the National Institute for the Blind, which has done so much and such valuable work for the blind, has appointed a prevention of blindness sub-committee. Its personnel is representative of all classes, lay and medical. Its chairman is Dr. C. G. Kay Sharp of Leeds. He commented upon the work of the new committee in his address at the conference of the Wales and Monmouthshire Regional Council of the Blind in Colwyn Bay. The National Institute for the Blind has done another good piece of work in preparing a report on blindness in India (Blindness in India, London, National Institute for the Blind, 1939. London, W.1). In a foreword Sir Michael O'Dwyer says there is no country in the world to-day where the affliction of blindness is so widespread as in India, nor where the ratio of blindness to population (350 millions) is so high. The totally blind are estimated at one and a half millions, the partially blind at three millions. Again, there is no civilized country in which so little is being done by the State, by public bodies, by voluntary associations (which hardly exist in India), and by private philanthropy to prevent blindness or to alleviate the lot of the blind. Among the reasons for the failure to tackle the problem are primarily the fatalism, common in the East, of regarding physical affliction as an act of God; the strength of the family and caste system, which, though producing many admirable results, limits the outlook and prevents the growth of a civic sense; the fact that nine-tenths of the population live in small and scattered villages or hamlets remote from the amenities of civilized life; and that only one-tenth are able to read and write. The report of the National Institute for the Blind includes an appeal to the Government of India. The International Association for the Prevention of Blindness has issued another number of its journal, which is a model for international publications, for it prints its matter in parallel columns of French and English. This issue deals with the recent annual session in London, at which Dr. Park Lewis of the U.S.A. was awarded the gold medal of the association for his great work in America. There are

reports on ophthalmia neonatorum in various countries; of particular interest are the notes from Dublin showing the excellent effect of prophylaxis. There are papers on eye diseases in workers by C. Coutela, and on lighting in schools by N. Bishop Harman. Of great interest is a note by H. Schröder on a small village called Titepec in Mexico, where all the inhabitants, indigenous and strangers, become blind. Investigations suggest that this blindness is caused by mosquitos which infect the people with a blinding worm of the genus *Onchocerca*."

Summer Graduate Course.—The eighteenth annual summer graduate course in ophthalmology and otolaryngology will be given in Denver, July 29 to Aug. 10, 1940. The list of guest teachers for the course in ophthalmology will be: Dr. Ramon Castroviejo, New York; Dr. John Hargreaves, Randolph Field, Texas; Dr. Placidus J. Leinfelder, Iowa City; Dr. Isaac S. Tassman, Philadelphia; Dr. Theodore L. Perry, Boston, and Dr. Sydney Walker, Chicago. The guest teachers for the course in otolaryngology will be: Dr. C. C. Bunch, St. Louis; Dr. A. J. Cone, St. Louis; Dr. W. J. Mellinger, Santa Barbara, Calif.; Dr. Harris Peyton Mosher, Boston; Dr. Samuel Salinger, Chicago, and Dr. Thomas Gooch Tickle, New York.

The fee for both weeks is \$50 and for one week only, \$30. This includes the cost of attending round table luncheons.

Obituaries

WEBB WILLIAM WEEKS, M.D.

1886-1940

After a lingering illness, Webb Weeks died at his home in New Canaan, Conn., on Jan. 10, 1940—a grievous blow to his friends and patients as well as to the medical profession at large.

He was born in Corry, Pa., on Oct. 24, 1886, the son of Frank Elmer and Eliza Cook Weeks. After his preparatory years at Corry High School, he entered Columbia University, graduating with the degree of Bachelor of Arts in 1910 and the degree of Doctor of Medicine in 1912. He then served a two year surgical internship at the Presbyterian Hospital in New York. After this, he began his training in ophthalmology at the New York Eye and Ear Infirmary. On the completion of this internship, he became associated with his distinguished uncle, Dr. John E. Weeks, until he entered military service.

His experience in the army was wide and varied, for after a short training period at Fort Sill, Okla., he went to France. Here he did active ophthalmologic duty both in evacuation and in base hospitals, and for this service he received two promotions. On his return to this country he was assigned to Fort McHenry, Md., where he worked with Dr. John M. Wheeler, gaining a large experience in plastic surgery. He was honorably discharged in July 1919, with the rank of major.

On resuming his work with Dr. John E. Weeks, his field of activity rapidly enlarged, and he soon became one of New York's leading ophthalmologists. His tireless energy, his keen clinical judgment and his surgical dexterity enabled him to profit to the fullest extent from this rare opportunity. A wise and experienced teacher had in him an apt and willing pupil.

He worked faithfully to the very last for the New York Eye and Ear Infirmary, serving it with true devotion and loyalty in almost every capacity. Starting as an assistant surgeon, he steadily earned promotions, until at the time of his death he was its executive surgeon. He was also director of the ophthalmic service of Bellevue Hospital and attending ophthalmologist to Beth Israel Hospital as well as consultant ophthalmologist to Rockefeller Institute, Norwalk (Conn.) General Hospital, Greenwich (Conn.) Hospital and Elizabeth A. Horton Hospital, Middletown, N. Y.

In addition to these clinical positions, he was professor of ophthalmology at New York University. His interest in undergraduate teach-

ing had been fostered by his predecessors, Dr. John E. Weeks and Dr. John M. Wheeler, under whom he had served as instructor and clinical professor before being appointed in 1928 to the professorship.

These responsibilities did not deter him from being a regular attendant at medical meetings. He served as chairman of the Eye, Ear, Nose and Throat Section of the New York State Medical Society in 1935 and of the Section of Ophthalmology of the New York Academy



WEBB WILLIAM WEEKS, M.D.
1886-1940

of Medicine in 1935 and 1936. He was president of the New York Ophthalmological Society in 1938. He took an active interest in the affairs of the American Academy of Ophthalmology and Otolaryngology, being a frequent lecturer at its instructional courses. His spirit of unselfishness was typically exemplified by this incident. Last October, in spite of great physical weakness and discomfort, he insisted on conducting one of these courses, doing it with his customary enthusiasm and thoroughness. He was also a member of the American Medical Associa-

tion, the American College of Surgeons, the American Ophthalmological Society and the Société française d'ophtalmologie.

He was not a prolific writer, but his articles were always cleancut, concise and practical. He was a contributor not only to the usual periodicals but also to Blumer's "Practitioner's Library of Medicine and Surgery" and to Berens' "The Eye and Its Diseases." He was actively interested in teaching operative surgery on the cadaver, giving much of his time and energy to this work. As an outgrowth of these courses, he prepared a manual on "Surgery of the Eye," in which he concisely portrayed the surgical procedures he had found useful.

He was a member of the University Club, the Country Club of New Canaan, the Wee Burn Club of Noroton, Conn., and the Phi Gamma Delta fraternity. He was also a member of the Quiz Medical Society and the Hospital Graduates Club, whose meetings he seldom missed.

In 1918 he married Ruth Thayer, of New Canaan, Conn., who with two children, John and Deborah, survives him. His home life was a happy one, for, although his professional activities demanded much of him, he always found time to hide away in New Canaan. A round of golf or a few sets of tennis were his chief diversions, with a detective story to top off a busy day.

In spite of all his accomplishments, a friend thinks first of him as a man, for to have known Webb was to have loved him. His genial nature, his generosity, his unselfishness and his high sense of integrity made him a delightful companion and a true friend. His boundless enthusiasm and great sense of fair play were ever present, both at work and at play. To those numbered among his friends his passing has left a void, never to be filled.

JOHN H. DUNNINGTON.

Obituaries

EUGEN VON HIPPEL

1867 - 1939

Eugen von Hippel, born in Königsberg, Germany, in 1867, was the son of Arthur von Hippel, professor of ophthalmology. So one might say that he was born with an aptitude for ophthalmology. But he was not only the son of a famous father. He acquired a place of honor among the ophthalmologists of the world by his own efforts. After being graduated from medical school, he had special training in internal medicine under Erb and in pathologic anatomy under Arnold. Then he turned to ophthalmology and became resident, assistant professor and associate professor in the ophthalmic hospital of the University of Heidelberg under the leadership of Theodor Leber. Thorough clinical training combined with scientific work was characteristic of this school. This principle remained with von Hippel all his life. One could fill many pages explaining all the work of Eugen von Hippel, but I want to emphasize only the most important of his scientific productions.

Scientifically, he was first of all a pathologist. Theodor Leber, Ernst Fuchs, Eugen von Hippel, Anton Elschnig and Joseph Meller are supposed to be the best ophthalmic pathologists in German-speaking countries. Eugen von Hippel himself always examined the rich material of enucleated globes in the ophthalmic hospitals in Heidelberg (1892 to 1909), Halle (1909 to 1914) and Göttingen (since 1914). In doing this, he not only illuminated the diagnosis of many cases but was stimulated to intensive research work on special topics. His work on siderosis bulbi (1893 to 1894), on glaucoma (1910) and on tuberculosis of the eyeball (1917 to 1918) are examples of profound anatomic and clinical studies. He never overestimated his own findings. When it appeared that his conception was wrong, he did not hesitate to admit it. For example, in the nineties he believed that tuberculosis, in addition to congenital syphilis, played a great role in the causation of keratitis parenchymatosa. He arrived at this opinion because of two observations: He had found epithelioid and giant cells on microscopic examinations in a case of keratitis parenchymatosa. He had also noted the presence of a disease of the joints, which was supposed to be tuberculous. When later in his own hospital, in the new era of syphilitic pathology, investigations made it evident that the foregoing criteria were

no proof of tuberculosis and that congenital syphilis was practically the only source of typical keratitis parenchymatosa, Eugen von Hippel did not hesitate to accept this idea.

Von Hippel not only was a pathologist but was occupied with experimental work. His investigations on the development of the normal eye and of malformations of the eye became the basis of all research in this field. He was the first who studied successfully the origin and inheritance of coloboma of the eye. He arrived at his conclusions by embryologic analysis of early stages, by experimental breeding and by the study of serial sections vertical to the fetal fissure (*Foetalspalt*). His three masterly monographs are on malformations of the eye.¹

In these monographs as well as in his work on the diseases of the optic nerve and on the cornea and in his many other articles, three points are especially admirable: the clear and concise language, the separation of certain and uncertain facts and the critical attitude he had toward his own findings and toward those of other investigators.

The name of von Hippel will always be connected with the disease which he first presented as a "very rare disease of the retina" and later described histologically. This condition is now known throughout the whole world as Hippel's disease (or Hippel-Lindau's disease). His opinion that the principal feature of the disease is an angiomatous tumor formation in the retina was first doubted but is now recognized as correct.

Also, his view based on Cushing's work on the belief that the choked disk is a pure pressure phenomenon was finally accepted. In many articles and lectures he pointed out that the tumor-choked disk has to be treated operatively by decompression, with or without removal of the growth in the brain. He felt deeply the responsibility of recommending a dangerous operation on the brain of a patient with choked disk and normal or nearly normal vision, but he felt that this was the correct procedure.

Without mentioning all the other articles of von Hippel, it must be said that he was in the true sense of the word a good clinician, familiar with all regions of the eye; that he examined every patient with great accuracy and knowledge, and that he was a good surgeon and a highly honored chief.

1. von Hippel, E., in von Graefe, A., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, Wilhelm Engelmann, 1901. von Hippel, E., in Schwalbe, E.: *Die Morphologie der Missbildungen des Menschen und der Tiere*, Jena, Gustav Fischer, 1909. von Hippel, E.: *Missbildungen*, in Henke, F., and Lubarsch, O.: *Handbuch der speziellen pathologischen Anatomie und Histologie*, Berlin, Julius Springer, 1928, vol. 11, pt. 2.

A few more words about the man. It is a great satisfaction for me, after having collaborated so many years with von Hippel, to speak of this often misunderstood teacher and friend.

Eugen von Hippel was by birthplace and nature an East Prussian. Duty was for him the superior law. An honest mind and reliability were to him indispensable. He seemed often to be cold and unapproachable, but this was only the expression of a certain kind of shyness and lack of flexibility. He had few friends, but very close ones; his best friend and companion, who took life so seriously, was his charming wife. More than most persons knew, he loved gaiety, humor, nature and music. It was a real pleasure to see this earnest man laughing so hard that the tears ran down his face or to observe him playing with his grandchildren. In those moments his longing for warmth and sunshine became obvious.

An outstanding side of his character was his feeling for justice and objectivity; because of this, he was a highly esteemed member of the faculty. His many pupils knew that they always had in him, in any emergency, a just advisor. On the other hand, he never was inclined to be a politician and to foster relations to procure favors for himself or for his pupils.

All in all, he was a real gentleman, a good ophthalmic surgeon, a successful investigator, a fine chief and a faithful friend.

It is a pity that because of sickness he was not able, after having resigned in 1934, to work out his plans and to give to his colleagues the results of his many unpublished studies.

JOSEPH IGRSHEIMER.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

THE OXIDASES AND REDUCTASES OF THE LENS. H. VON EULER, H. HELLSTROEM, F. SCHLENK and G. GUENTHER, *Arch. f. Ophth.* 140: 116 (Feb.) 1939..

Cattle and rat lenses were examined and found to contain all enzymes necessary for glycolysis, whereas some of the most important oxydases, like cytochrom, were missing. The well known presence of large amounts of ascorbic acid and of sulfhydryl compounds (glutathione) in the lens was confirmed.

P. C. KRONFELD.

Conjunctiva

BITOT'S SPOTS IN CEYLON. L. NICHOLLS and A. NIMALASURIYA, *Lancet* 1: 1432 (June 24) 1939.

Nicholls and Nimalasuriya encountered a condition in the bulbar conjunctiva of several hundred malnourished children and other persons in Ceylon, which they refer to as Bitot's spots. The pathologic changes, as seen by them, are of a definite type but do not conform to some of the descriptions given by other observers. They have never seen Bitot's spots extending to the cornea, for they do not regard the changes in the cornea which occur in keratomalacia as extensions of Bitot's spots; and the spots in the great majority of their cases have in no way resembled foam. Bitot stated that all his patients had night blindness, but this has not been present in most of the authors' patients. Bitot's spots tend to be chronic, and it is necessary to give large doses of fish liver oils or concentrated preparations of vitamin A to clear them. The authors state that there may be two forms of xerosis, or keratinization, of the bulbar conjunctiva, an acute form in which the changes take place rapidly, forming loose accumulations of degenerative epithelium, which appear foamy, and a chronic form in which the accumulations of epithelium take place more slowly and are more compact. If this is so, possibly the first form is more likely to occur as a rare condition in countries in which diets deficient in vitamin A are not in common use, and the second form, as in the authors' patients, will be seen in countries in which the inhabitants have become more or less biologically adapted to diets deficient in this vitamin and consequently their reactions to the deficiency are more chronic.

J. A. M. A.

CONJUNCTIVITIS CAUSED BY A FOREIGN WOOD, MAKORÉ: REPORT OF A CASE. DEFRANCE, *Bull. Soc. d'ophth. de Paris* 51: 90 (Feb.) 1939.

A cabinetmaker had gotten some sawdust in his right eye, which caused redness and rhinorrhea with tearing for some two weeks. On

the second occasion the sawdust caused a persistent irritation, redness and lacrimation for two or three weeks. After a third attack examination showed normal lids, but the bulbar conjunctiva was markedly vascularized. The cornea was entirely normal, as were the iris and lens. The ocular tension was 18. There was no adenopathy. A smear of the bulbar conjunctiva showed an absence of organisms. Zinc sulfate, used locally, did not help the condition. Experimentation with wood powder in a rabbit's eye produced a marked reaction. Defrance concludes that makoré is more irritating than ordinary wood powder. Makoré wood was found to be impregnated with zinc, copper and mercury in fine powder form. It is felt that these metals account for the reaction. Whether the mechanism is entirely one of mechanical irritation or sensitivity could not be decided.

L. L. MAYER.

Comparative Ophthalmology

THE EYE OF ARGYROPELECUS HEMIGYMNUS. F. CONTINO, Arch. f. Ophth. 140: 390 (April) 1939.

Argyrops leucostictus hemigymnus ("half-naked silver hatchet") is a small deep-sea fish which is found in the Straits of Messina. The author has studied about 100 specimens, representing all stages of development. The adult fish is from 3 to 4 cm. long and possesses so-called telescopic eyes, that is, eyes of approximately cylindric shape. The retina is situated at the base of the cylinder. The cornea contributes the lateral portion of the wall and the convex top of the cylinder. Only the latter is used for seeing. These eyes are inserted vertically in the head of the adult fish, so that most of the animal's field of vision lies above its head. The punctum remotum is situated 20 mm. above the eyes. The lens is spherical and protrudes markedly into the anterior chamber. The action of the retractor lentis has a negative accommodative effect and moves the punctum remotum to a point about 1 meter from the eyes. The retina contains only rods. The cornea is apparently impermeable to ultraviolet rays. The animal's eye is well equipped for the perception of the low intensities of light which prevail in the deeper strata of the sea.

P. C. KRONFELD.

Cornea and Sclera

RECURRENCE OF SYPHILITIC INTERSTITIAL KERATITIS. E. DALSGAARD-NIELSEN, Acta ophth. 17: 38, 1939.

The incidence of recurrence of attacks of interstitial keratitis has been variously reported at from 5 to 17 per cent. The author is convinced that many of these recurrences are spurious; that is, they do not represent a real recurrence of keratitis but rather a recurrence of iridocyclitis, or a reengorgement of the deep corneal blood vessels as a result of irritation.

A reexamination of 173 patients who had formerly had interstitial keratitis failed to reveal any instance of true recurrence.

O. P. PERKINS.

BLUE SCLERA. H. L. JENSEN and K. K. ORTMANN, *Ugesk. f. læger* 101:275 (March) 1939.

Jensen and Ortmann, reporting 2 cases of Lobstein-Eddow's disease, osteopsathyrosis idiopathica, state that in the disorder blue scleras, a tendency to fractures and otosclerosis constitute a triad. As seen in their second case, blue scleras are not always accompanied by the tendency to fractures. There is generally a proportionality between the degree of coloring and this tendency. All authors agree that blue scleras are the dominating symptom. The color has been observed in 5 generations and established in 50 descendants of 1 person. A certain intensity of the coloring, a familial character and the combination of osseous and auditive stigmas in at least 1 member of the family are necessary for a positive diagnosis. The abnormal fragility of the bones depends on insufficient periosteal ossification coincidentally with inability of the bone tissue to take up calcium. The patients may become greatly deformed; in cases of extreme involvement they are referred to as "men of glass." The more underlying cause of the symptom complex is unknown. While in 1 family the disorder has appeared only in 20 male members, as a rule both men and women are affected. Transmission is probably dominant. The disorder may skip several generations, to reappear as the result of exogenic (disease) or endogenic factors (puberty or gravidity).

J. A. M. A.

Experimental Pathology

ARTIFICIAL ACHROMATOPSIA PRODUCED BY DAZZLING. Y. LEGRAND and E. GEBLEWICZ, *Bull. Soc. d'opt. de Paris* 51:171 (March) 1939.

It has been well known for a long time that color vision is less stable in lateral than in foveal vision. Apparently the saturation of a light impinging on the periphery of the retina decreases with time. That effect becomes considerably more marked if the light, instead of being continuous, is interrupted at a frequency of from forty to fifty per second. Experimenting with monocular vision, the authors have investigated a region 15 degrees from the fovea. If a dazzling light is used, the recognition of colors is reduced to a minimum. The implication theorized is the absence of cones and predomination of rods. One may think that the phenomenon of synchronization may so affect the nerve as to permit only uncolored stimuli to reach the cortex. The authors believe that the explanation is still unfounded but that the phenomenon proves that in all cases the lateral portion of the retina possesses special properties at high levels of brilliancy.

L. L. MAYER.

ACTION OF MINERAL DUSTS ON CONNECTIVE TISSUE, AS STUDIED WITH THE CORNEAL MICROSCOPE. A. POLICARD and J. ROLLET, *Compt. rend. Soc. de biol.* 130:954, 1939.

Traces of sterilized mineral dusts were inserted by a minute oblique incision in rabbit corneas which had been anesthetized with cocaine

hydrochloride. Coal dust incited no reaction, the carbon particles remaining at all times sharply delineated. Quartz and other silicas (opal, sand and fullers earth) produced a mild, slowly developing interstitial reaction, evident by the twelfth day as a whitish halo, consisting of cellular infiltration. With mica and sericite, the reaction was slight and transient. Permutite and cobalt arsenate, however, occasioned an early and severe reaction consisting of a grayish halo about the deposit and edema of the stroma and overlying epithelium.

J. E. LEBENSOHN.

General Diseases

DYSOSTOSIS CRANIO-FACIALIS (CROUZON). A. C. KRAUSE and D. N. BUCHANAN, *Am. J. Ophth.* 22: 140 (Feb.) 1939.

Krause and Buchanan discuss malformations of the head, their classification and the symptoms, signs, prognosis and treatment of cranio-facial dysostosis. They report a case in a 2½ year American boy and give the following summary:

"Reported in this article is a case of dysostosis cranio-facialis or Crouzon's disease with typical symptoms and confirmed by roentgen-ray examination. There is no apparent record of this disease in the American literature. In spite of operative decompression of the cranium and right orbit there is no reason to believe the prognosis will be favorable for either the eye, mentality, or life."

W. S. REESE.

General Pathology

THE ERYTHROCYTE-PLASM CHLORIDE RATIO IN OCULAR DISEASES. D. MICHAIL and P. VANCEA, *Compt. rend. Soc. de biol.* 130: 1043. 1939.

A definite hypochloremia, indicative of a tendency to alkalosis, is found in glaucoma, detachment of the retina, optic neuritis, atrophy of the optic nerve, opacities of the vitreous and phlyctenulosis; an increased chloride ratio is noted in interstitial keratitis, sympathetic ophthalmia and disciform keratitis.

J. E. LEBENSOHN.

Lids

A CASE OF ALEUKAEMIC LYMPHOSIS INVOLVING THE UPPER LIDS. WITH PATHOLOGICAL FINDINGS. F. TOOKE, *Brit. J. Ophth.* 23: 444 (July) 1939.

A man aged 65 was admitted to the hospital with a pendulous right lid. A tumor was presented. The skin of the lid was not discolored and was movable over the underlying tumor. The palpebral conjunctiva was thick and lusterless and a pinkish gray, resembling somewhat sodden blotting paper. The upper lid of the left eye presented prac-

tically the same features. Except for a moderate degree of chemosis about the accessory lacrimal glands, the globes appeared normal. There was a uniform adenopathy. Roentgenograms showed marked enlargement and increased density involving the roots of both lungs. This may have been due to hypertrophied glands. Biopsy of the tumors and of the posterior cervical glands gave a picture corresponding to that found on the borderline between lymphatic leukemia and a relatively mature form of lymphosarcomatosis. The author is inclined to classify the disease among the rather immature forms of lymphatic leukemia. The blood picture showed evidence of marked anemia, with a tendency for the cells to be somewhat larger than normal and to have poor regenerative ability. The white blood cells showed deficiency in the number of the myeloid elements, with some immature forms. The lymphocytes were increased; the increase appeared to be due to the presence of immature lymphoblastic cells, corresponding to those to be met with in the lymph glands.

The concluding paragraph of the article contains the following statements:

"This case, therefore, is one of a systemic lymphoid proliferation, which apparently from the progressive changes in the blood picture has involved the bone marrow, spleen, and lymph glands of the patient, as well as in an unusual manner, the eyelids. The proliferation is of a comparatively active form, and falls in its intensity and character between that met with in lymphatic leukaemia and that in lymphosarcomatosis. For this reason it is best classified as a leukaemic form of lymphosis, with unusual involvement of the eyelids and with an activity of cell proliferation bordering on the lymphosarcomatoses."

W. ZENTMAYER.

Methods of Examination

A COMPARISON OF LENS AND SKIASCOPE METHODS IN RETINOSCOPY WITH UNDILATED PUPILS. W. J. B. RIDDELL, *Brit. J. Ophth.* 23: 387 (June) 1939.

The author gives the following summary of his article:

"A useful type of skiascope has been re-described.

"It has been used for hospital refraction work for over thirty years, and has proved a valuable time saving device.

"Figures provided by two hundred and fifty cases in which both trial frame and skiascope were used have been compared.

"A high degree of correlation was found.

"It is suggested that this device might be employed in the construction of refraction curves from unselected samples of the population.

"The skiascope readings tended to be lower (i. e. more myopic) than the lens methods."

W. ZENTMAYER.

ANALYSIS OF THE JUDGMENT OF RELATIVE POSITION: PRELIMINARY COMMUNICATION. P. C. LIVINGSTON, *Brit. J. Ophth.* 23: 540 (Aug.) 1939.

The object of this paper is to bring into the foreground certain features of binocular vision which do not appear to have received full recognition and which, studied more fully, appear to have a close association with depth perception. Considered here, the term "depth perception" means the highest stratum of spatial recognition. A rotating depth perception apparatus is described. The test is based on the three pins of Helmholtz. The observer, at a distance of 11 feet (3 meters) from the pins, is asked to reset, by means of a cord, the middle pin in a position which he feels will make all three pins equidistant from him. Four perimeter positions are employed. It is believed by this procedure that the two visual characters are assessed, namely: (1) the sensitivity of the retinas to stimulation at disparate points, and (2) the psychovisual response to such stimuli, which calls for interpretation varying in acuteness according to the rotated position of the pins.

A second apparatus, consisting of a rotating stereogram and pictures, is used.

Experiments with these charts have revealed already that the majority of observers experience alternate vision and that there are marked variations in the quality alternation. W. ZENTMAYER.

Neurology

PAPILLITIS AND PAPILLEDEMA IN MULTIPLE SCLEROSIS. S. SUGAR, *Am. J. Ophth.* 22: 135 (Feb.) 1939.

Sugar comments on the polymorphic manifestations of multiple sclerosis and the paucity of cases showing papillitis and papilledema. He gives the following summary:

"Two cases of choked disc occurring in multiple sclerosis are presented together with a review of the literature.

"In any case of choked disc with marked impairment of vision multiple sclerosis should be considered in the differential diagnosis.

"Recognition of the occurrence of this condition may help avoid unnecessary neurosurgical procedure." W. S. REESE.

GLIOMAS OF THE PONS. B. J. ALPERS and J. C. YASKIN, *Arch. Neurol. & Psychiat.* 41: 435 (March) 1939.

Most pontile gliomas occur in children. Of the 11 tumors in this series, 8 occurred in children under 11 years of age. In most of the cases the illness began with diplopia and headache. The most striking of all the signs of pontile glioma were the paralyses of the cranial nerves, which were present always in greater or lesser degree. The abducens nerve was most frequently affected. R. IRVINE.

A SIGN OF FACIAL PALSY. R. WARTENBERG, Arch. Neurol. & Psychiat. 41: 586 (March) 1939.

Decrease of vibration in the upper lid felt by the examiner's palpat-ing finger, which exerts upward pressure on the upper eyelid as the patient closes the eye, constitutes the finest and most reliable sign of facial palsy.

R. IRVINE.

FOVILLE'S SYNDROME: RECORD OF A CASE. J. R. MUTCH, Brit. J. Ophth. 23: 225 (April) 1939.

A man, aged 56, complained of failing vision and watering of the left eye. The positive symptoms were: paralysis and wasting of all the muscles of the left side of the face; left internal strabismus; loss of conjugate movement of each eye to the left; ptosis on the right; unequal pupils; horizontal nystagmus when the patient looked to the right; slurring of speech; sclerosis of the retinal vessels, and unsteadiness of gait with a tendency to fall to the left.

Mutch gives an exhaustive analysis of the symptoms, a description of the anatomy of the pons and its blood supply and a discussion of the relation of the symptoms to the nuclei. On the basis of the evidence supplied, the sudden onset, the nonprogressive nature of the lesion, the state of the retinal arteries and the fact that the lesion is confined to such a small area of the pons, the author made a diagnosis of thrombosis of a median branch of the basilar artery.

The article is illustrated.

W. ZENTMAYER.

Ocular Muscles

CONGENITAL ABDUCENS PARALYSIS. C. APPLE, Am. J. Ophth. 22: 169 (Feb.) 1939.

Apple reviews paralysis of the abducens nerve as to cause, preponderance on the left side and important objective findings, namely, restriction of abduction, restriction of adduction, retraction, oblique upward and downward movements, protraction, convergence insufficiency and torsion movements. He reports 3 cases from a total of 15 that were observed at the ophthalmic clinic of the University of Illinois College of Medicine in the past ten years. The patients in these 3 cases were personally observed and operated on to determine the underlying anatomic condition and to correct the strabismus.

W. S. REESE.

Parasites

MYIASIS DUE TO OESTRUS OVIS. TEMPLE, HARANT and VIALLEFONT, Arch. Soc. d. sc. méd. et biol. de Montpellier 20: 26 (Feb.) 1939.

Oestrus ovis habitually lays its eggs in the eyes and nostrils of sheep, goats and dogs and only rarely in the human eye. Two cases of myiasis due to Oestrus ovis are reported in human beings. In 1 case a soldier complained of having something in his left eye since the previous evening. With the slit lamp one could just discern the movements of an

almost transparent body. Only the darker hooks of the larva and the particles in the digestive tract were perceptible. With the aid of the slit lamp the larva was removed without difficulty with the patient under local anesthesia and was examined under the microscope. It was 1.5 mm. in length and 0.4 mm. in width. In the second case the patient, a man, felt that an insect had entered his left eye the evening before. Examination revealed six larvae. Clinical manifestations are limited to tingling and a slight conjunctival reaction.

J. E. LEBENSOHN.

Pharmacology

THE EFFECT OF THE MELANOPHORIC HORMONE IN NORMAL PERSONS AND IN THOSE WITH RETINITIS PIGMENTOSA. G. BASILE, *Ann. di ottal. e clin. ocul.* 67:412 (June) 1939.

The author investigated the effect on light sense and dark adaptation of an extract of the intermediary lobe of the pituitary which was known to produce expansion of the melanophores in the skin and retina of frogs. The literature, in part conflicting, is discussed. One cubic centimeter of the solution employed contained 20 to 25 units, as determined by Jores. During light adaptation of ten minutes, several drops of the solution was instilled in the right eye of the subject, the left being used as the control. The light sense was tested with the adaptometer of Engelking after dark adaptation every five minutes until the end of a forty-five minute period. Twenty normal subjects were first employed. A definite acceleration in dark adaptation with an increase in the luminous threshold was observed in the eyes receiving the hormone.

A similar technic was carried out in 6 patients with retinitis pigmentosa showing marked night blindness. In all of these cases a definite rise in the luminous threshold and an increase in light sense were observed. In a third series, 7 patients with retinitis pigmentosa were given the hormone by subcutaneous injection, adaptation being tested before and during treatment. Injections of 0.5 to 1 cc. were given every two to four days and from three to nine injections. Marked increase in the light sense was observed after treatment in 5 of these patients, the vision and fields showing, on the contrary, no improvement. The response in 2 cases was less satisfactory, both of these cases showing a marked degree of night blindness. Further investigations are proposed to determine whether prolonged use of the hormone will affect the course of the disease. The mode of action on the retinal elements has not been determined.

S. R. GIFFORD.

THE INFLUENCE OF RETROBULBAR INJECTIONS ON THE TENSION OF THE EYE. N. MEDVEDIEV and L. SATZ, *Vestnik oftal.* 14:102, 1939.

The authors gave retrobulbar injections of procaine hydrochloride and epinephrine hydrochloride in a solution of a 0.5 per cent to 18 rabbits and 29 patients with various diseases. There was a decrease in tension in the majority of the animals and patients, but in 6 patients an increase of the tension was observed after the injections, particularly

in the eyes with increased tension. Medvediev therefore thinks that retrobulbar injections should be given cautiously in cases of primary and secondary glaucoma. The increase of tension might be due to the needle and also to the mechanical pressure of the liquid.

O. SITCHEVSKA.

The Pupil

TONIC PUPILS AND ABSENT TENDON REFLEXES (ADIE'S SYNDROME).
A. VEASEY SR., Northwest Med. 38: 204 (June) 1939.

Veasey reviews the literature of Adie's syndrome and reports 2 cases of his own. The patient in the first case was an unmarried girl aged 18. She complained of the pupil of the right eye being larger than that of the left eye. A photograph showed that the condition was present at 9 years of age. Vision was 6/5 in each eye. The media and fundus were normal. The behavior of the pupils was typical of the syndrome. The pupils responded promptly to mydriatics and miotics. There was no Romberg sign; both knee jerks and elbow jerks were normal, but both ankle jerks were absent. The laboratory tests and physical examination otherwise gave negative results.

The patient in the second case was an unmarried woman aged 24. The pupil of the left eye had been dilated at least six months. The history was irrelevant. Examination revealed nothing of importance. The pupillary reactions were those of the syndrome. The pupils reacted to miotics and mydriatics. The visual acuity was normal. The media and fundus were normal. The knee, elbow and ankle jerks were absent on both sides.

W. ZENTMAYER.

Physiology

THE INFLUENCE OF CENTRAL NERVOUS SYSTEM ON THE PIGMENT
MIGRATION IN THE RETINA OF THE FROG. H. M. BURIAN, Am. J.
Ophth. 22: 16 (Jan.) 1939.

Burian gives the following summary of his investigations:

"Experiments with strychnine and naphthalene have shown that the retinal pigment of dark-adapted frogs shows light position when the animals are poisoned with strychnine, and that in light-adapted frogs poisoned with naphthalene the retinal pigment assumes dark position.

"From this the author concludes that there is a regulating influence of the central nervous system on the position of the retinal pigment.

"In frogs, in which the optic nerve on one side was severed, the drugs did not influence the eye operated upon. The author concludes that the pathway through which the central nervous system exerts its influence must be located in the optic nerves, supporting Engelmann's theory of the existence of centrifugal, retinomotor fibers in the optic nerve.

"The position of retinal pigment appears to be influenced by the following factors: (1) the basic tonus of the central nervous system; (2) the 'light tonus' of the central nervous system; and (3) local humoral conditions due to the influence of light and various general metabolic factors."

W. S. REESE.

Retina and Optic Nerve

PATHOGENESIS OF THROMBOSIS OF THE CENTRAL RETINAL VEIN AND OF CONSECUTIVE GLAUCOMA. P. WEINSTEIN, *Brit. J. Ophth.* 23: 396 (June) 1939.

Weinstein found that glaucoma developed in 23 of 50 persons with thrombosis of the central vein of the retina. The author gives the following summary:

"Thrombosis of the central retinal vein occurs in individuals of advanced age, whose blood-pressure is high (180 mm. of mercury or above), the amplitude of their tonoscillograms exceeding 15 mm.

"The histological examination of the central retinal vein discloses its walls to have thickened, its endothelium displaying hyaline degeneration and proliferation, the adventitia disclosing hyperplasia of connective tissue.

"In such cases thrombosis of the central retinal vein is followed by glaucoma, its main branch being obliterated.

"Glaucoma forms in such cases by accumulation of pathological products of altered metabolism (carbonic acid, lactic acid, acetic acid, citric acid, etc.), the vitreous body swelling gradually by their influence. Under the normal physiological conditions, hydration corresponds to 98.5 mm. of water, and under the influence of lactic acid augments to 166.0 mm.

"X-ray irradiative treatment of hemorrhagic glaucoma is indicated."

W. ZENTMAYER.

THE DISEASE OF LAURENCE-MOON-BARDET-BIEDL (RETINITIS PIGMENTOSA, POLYDACTYLISM, ADIPOSEGENITAL DYSTROPHY AND INTELLECTUAL DEFICIENCY). C.-I. URECHIA, P. VANCEA and L. DRAGOMIR, *Ann. d'ocul.* 176: 274 (April) 1939.

This rare and interesting malady was reported by Laurence and Moon for the first time in 1866 in 4 sisters, in whom there was found retinitis pigmentosa associated with retarded physical and intellectual development. Bardet, in an excellent thesis in 1920, entitled "The Syndrome of Infantile Obesity with Polydactylism and Retinitis Pigmentosa," described the condition and drew attention to the fact that the malady seemed to still remain unknown. Two years later, in 1922, Biedl took up the study of the disease and added to the syndrome mental deficiency. He pointed out that in 2 of his cases there were present posterior cortical cataract and anal atresia. To date there have been about 150 cases reported.

The case is reported of a youth aged 21 whose father suffered from mental debility but was without any malformation. The case described is a classic one of the disease, which up to the present has been seen only in white persons.

There are five illustrations and five pages of bibliography.

S. H. McKEE.

COAT'S RETINITIS. V. SPADAVECCHIA, *Ann. di ottal. e clin. ocul.* 67: 321 (May) 1939.

The author reports a case of bilateral retinitis haemorrhagica externa occurring in a man of 40. The visual disturbance had begun one and

one-half years before he was seen by the author. Vision was reduced to 2/10 in the right eye and to perception of hand movements in the left eye. The fundus changes are well illustrated and showed the typical formation of connective tissue in the retina with dense bands connecting various retinal areas, many containing newly formed vessels. These were especially marked in the left eye, in which the disk was completely covered. The veins were large and tortuous, and the small branches were surrounded by tissue resembling glia. In both eyes a large subretinal mass was seen occupying a large part of the fundus and accounting for 9 diopters of hyperopia in the right eye and 20 diopters in the left, as determined by retinoscopic examination. General examination showed numerous telangiectases of the skin, moderate hypertension and slight enlargement of the liver and spleen. Microscopic study of the capillaries showed a moderate loss of tone in the venous branches of the capillaries. Tuberculin tests gave slightly positive reactions. The signs indicated a tendency to generalized venous stasis. The author believes that this abnormal condition of the veins may be of causative importance in relation to the ocular changes. The literature is discussed.

S. R. GIFFORD.

TREATMENT OF RETINITIS PIGMENTOSA WITH INTRAMUSCULAR INJECTIONS OF COD LIVER OIL. V. FILATOV and E. VERBITZKAYA, *Vestnik oftal.* 14: 21, 1939.

Six patients suffering from retinitis pigmentosa were treated with intramuscular injections of pasteurized cod liver oil. In 3 patients the injections produced a marked and quick improvement of the function of the retina, disappearance of the night blindness and improvement of dark adaptation, visual fields and visual acuity. In 2 patients the treatment yielded moderate results, and in 1 there was no improvement. Though the period of observation is short (from one to four months), the authors think it worth while trying this treatment on a large number of patients.

Filatov believes that the action of the cod liver oil depends not only on vitamin A but on the products of disintegrated liver tissue. In order to verify this, two small pieces of conserved human liver were implanted under the skin of a patient with retinitis pigmentosa. The vision improved from 0.3 to 0.4, the visual fields increased about 10 degrees and the dark adaptation improved. The histories and visual fields of the patients are presented in the article.

O. SITCHEVSKA.

Trachoma

THE ÆTIOLOGY OF TRACHOMA. F. H. STEWART, *Brit. J. Ophth.* 23: 373 (June) 1939.

Stewart gives the following summary of his views concerning the etiology of trachoma:

"Trachoma is caused by the virus described in 1907, the most conspicuous form of which is the Prowazek-Halberstaedter inclusion body or P. K. A free extracellular stage also exists resembling in appearance

a polymorph bacterium. Inclusion bodies can be found in all cases of trachoma if examination is repeated. Allied diseases are paratrachoma (inclusion blennorrhoea and swimming bath conjunctivitis), psittacosis and granuloma inguinale, the parasites of which resemble that of trachoma. Reports on filtration through collodion membranes impermeable to bacteria, of 0.6 and 0.7μ a.p.d. are contradictory (1) that the filtrate is infective (2) that it is not so. The virus can be concentrated on the upper surface of such membranes. It has not been grown in any form of culture or on the chorio-allantoic membrane. Animals which can be infected with certainty in the conjunctiva are baboons and grivet monkeys. The virus can survive in the testicle of rabbits and guinea-pigs, and it is claimed will multiply in the intestines of a louse. The statement that trachoma is normally carried by lice needs confirmation. There is some experimental evidence that it is not carried biologically by lice or flies. Infection is spread by direct contact and may be carried mechanically by flies for a short time only, since the virus is killed by drying."

The article is illustrated.

W. ZENTMAYER.

Therapeutics

ACTION OF VITAMIN B_1 IN CERTAIN OCULAR DISEASES. G. CARLEVARO, *Ann. di ottal. e clin. ocul.* 67: 355 (May) 1939.

The author has attempted to group a number of corneal conditions, including herpes zoster, herpes simplex, disciform keratitis, recurrent erosion and others, under the heading of neurotrophic corneal diseases. In a series of such cases crystalline vitamin B_1 was administered intravenously and ethylmorphine hydrochloride locally. In 6 cases of herpes simplex of the dendritic type, and in 1 of superficial punctate keratitis successful results were obtained. In another case of dendritic ulcer recurrences were noted in spite of treatment; there were also recurrences in a case of unusually severe diffuse keratitis. No result was obtained in a case of herpes zoster with iridocyclitis or in a case of disciform keratitis. In another case of disciform keratitis some improvement occurred under treatment. In only 1 case was there definite evidence of a vitamin-deficient diet, although a history of alcoholism, influenza and tuberculosis in several cases offered a possible explanation of a deficiency.

Vitamin B_1 was employed in a second series of cases of diseases of the optic nerve. In 3 cases of toxic amblyopia due to tobacco and alcohol good results were obtained, while no effect was noted in cases of retrobulbar neuritis due to multiple sclerosis and syphilis.

To determine the effect of vitamin B_1 on regeneration of nerve tissue, the author compressed the sciatic nerve of rabbits, treated alternate animals with vitamin B_1 and studied sections of the nerves after various intervals of one to sixty days. Some evidence was obtained indicating a more rapid repair of damage to the myelin sheaths in the animals receiving vitamin B_1 . The literature is discussed, and a bibliography is included.

S. R. GIFFORD.

TREATMENT OF ACNE ROSACEA WITH EXTRACT OF THE CORTEX OF THE ADRENAL GLAND. R. THIEL, *Klin. Monatsbl. f. Augenh.* 102: 394 (March) 1939.

An ointment containing zinc ichthammol has yielded good results in keratitis caused by acne rosacea but has failed in many cases, some of which were reported by Claussen and by Wilhelm. Thiel refers to contributing disorders in acne rosacea, such as subacidity, constipation, anemia and endocrine dysfunction, especially prior or during the menopause, and mentions Wadel's satisfactory experience in cases of acne rosacea with hormones of the hypophysis and of the cortex of the adrenal gland. Two cases of Thiel's own observation are reported in which acne rosacea of the face with recurring keratoconjunctivitis resisted various treatments. Both patients, a man aged 43 and a woman aged 51, recovered promptly after injections of an aqueous solution of extract of the cortex of the adrenal gland prepared after an especial method. The man received eight injections and the woman fifteen. Equally good results were obtained in 8 other patients. No untoward symptoms developed, except in 1 patient, in whom small abscesses formed at the site of the injection; they were attributed to local allergic reaction. The preparation is manufactured by the I. G. Farbenindustrie, A. G., Germany.

K. L. STOLL.

Toxic Amblyopia

INVERSION OF THE VISUAL FIELD FOR COLOR IN AMBLYOPIA DUE TO QUININE. C. DRACONTAIDIS, *Ann. d'ocul.* 176: 437 (June) 1939.

In the foreign literature, especially in the Greek, many cases of quinine amblyopia are recorded in which there is noted a concentric shrinking of the limit of the visual field for plain and colored lights.

The writer has noted at the ophthalmological clinic at the University of Athens a considerable number of cases of amblyopia due to ingestion of quinine in which detailed examination of the color fields was made. In 8 of the cases of the previous year an inversion of the limit of the visual field for color was found. Examination showed a normal disposition of the limits of the visual fields for different colors, but a definite inversion of these was found. The 8 cases are described in considerable detail, with comments on each and the visual fields in each case. A bibliography accompanies the article.

S. H. McKEE.

Uvea

A NOTE ON CHRONIC IRIDO-CYCLITIS, WITH SPECIAL REFERENCE TO THE SARCOIDOSIS OF BOECK. R. KEMP, *Brit. J. Ophth.* 23: 455 (July) 1939.

Kemp examined 25 patients with chronic iridocyclitis to establish to what extent such persons were actually suffering from generalized sarcoidosis. In all the cases the condition was chronic and untractable, and in none had the etiologic agent been found.

Based on this study, the author concludes:

"1. The incidence of non-specific chronic irido-cyclitis seems to fall into three age/sex groupings.

"2. Apart from this, present day clinical methods are unable to link the illness with any of the known aetiological causes. It may therefore be a disease *sui generis*.

"3. It follows a chronic progressive course, is not amenable to treatment and the visual prognosis is poor.

"4. Sarcoidosis is not a cause in the majority of instances."

W. ZENTMAYER.

CHOROIDITIS AREATA. K. SVEINSSON, *Acta ophth.* 17:73, 1939.

The author reports 4 cases of a fundus lesion which Professor Rønne has called choroiditis areata. The condition is thought to be the result of a congenital anomaly in the development of the pigment epithelial layer and of the choroid. It is bilateral, with a tendency to symmetry. A glistening white area of choroidal atrophy surrounds the disk, and similar tongue-shaped areas, with scalloped borders, radiate toward the periphery. The maculas are not involved, nor are there other pathologic symptoms or signs. Black and white drawings illustrate the 4 cases.

O. P. PERKINS.

Society Transactions

EDITED BY W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

DAVID WEBSTER, M.D., *Chairman*

ROBERT K. LAMBERT, M.D., *Secretary*

Dec. 18, 1939

Bilateral Subconjunctival Tumor. DR. ALBERT V. SARADARIAN.

Painless, extensive, subconjunctival salmon-colored tumors developed in the upper halves of both globes of McC., a white man aged 59, whose general health was good. He entered the Jersey City Medical Center Clinic and Hospital for observation and treatment. Examination revealed chronic sinusitis, severe caries, a faintly positive Mantoux test, an increased basal metabolic rate, right submental adenopathy, bilateral axillary and inguinal adenopathy, a shift to the left of juvenile non-segmented polymorphonuclear cells, mild secondary anemia and leukocytosis; biopsy showed numerous undifferentiated mast cells.

DISCUSSION

DR. JOSEPH IGRSHEIMER, Istanbul, Turkey: About fifteen years ago I encountered a case similar to the one reported by Dr. Saradarian. The patient was a woman of about 50 who was suffering from aleukemic leukemia.

Vaccinal Disciform Keratitis Following Accidental Inoculation of the Eyelid. DR. CHARLES A. PERERA.

This paper will be published in full, with discussion, in a later issue of the ARCHIVES.

Ocular Sensitivity to Nupercaine. DR. CHARLES A. PERERA.

This paper will be published in full, with discussion, in a later issue of the ARCHIVES.

Vitamin A and Vision. SELIG HECHT, PH.D., Columbia University.

Measurements of dark adaptation record the changing amount of light required for the threshold of vision during a stay in the dark subsequent to exposure of the eye to light. In general, the course of dark adaptation shows two portions. The first is an immediate and rapid decrease in threshold, which is associated with cone function; the second is a later and slower decrease in threshold, which is associated with rod function. These two parts of the course of dark adaptation may be demonstrated in a variety of ways, depending on the differences

between the rods and cones in their retinal distribution and in their sensibility to the spectrum.

The precise course of dark adaptation is influenced by several different factors: by the duration, intensity and color of the preadapting light and by the size, retinal location, color and duration of the test light used for measuring the threshold. In other words, the measurement of dark adaptation is not a haphazard process but must be made under rigidly defined conditions in order for the results to have precision and meaning.

Dark adaptation is an expression of the accumulation in the eye of photosensitive substances, such as visual purple in the rods and visual violet in the cones. This accumulation of sensitive materials is determined among other things by the vitamin A content of the diet, since night blindness due to dietary deficiency has been cured by the addition of vitamin A to an inadequate diet. In addition, vitamin A is formed in the retina when it is bleached. Thus there exists in the retina a chemical system in which vitamin A is both precursor and product of the principal photosensitive materials.

In terms of this chemical relation, efforts have been made to use measurements of the visual threshold as diagnostic signs for the vitamin A state of the body, particularly in cases of subclinical avitaminosis A. The results have been conflicting and have shown that enthusiasm is no substitute for adequate knowledge of dark adaptation and vision or for precise methods of measurement.

To establish critical information in this respect, an apparatus was designed which controls all the factors known to influence the progress and extent of dark adaptation. With this apparatus under standard fixed conditions, a survey was made of a reasonably large and normal group of persons in order to establish the norms for the various characteristics of dark adaptation. The most significant of these turned out to be the final threshold for cone vision, the final threshold for rod vision and the time for the point of transition from cone to rod function. Sex has no influence on any of these properties. Age increases the threshold both of the cones and the rods, though the cone-rod transition time is unaffected.

Experiments with 17 normal young men on controlled diets showed that most subjects respond immediately to a drastic reduction in the vitamin A content of their diet. There is considerable individual variation, but the rise in threshold is great and unmistakable and in most cases immediate. In 3 cases the rise was slight to begin with, and a rapid rise showed up only after two months of continued deficient diet.

Recovery from a deficient diet as measured by the visual threshold was nearly always slow. Ingestion of large single doses of vitamin A (100,000 to 500,000 U. S. P. units) produced variable reductions in the threshold, but the reductions were never large, and the dramatic results achieved by other workers have not been duplicated. The return to a normal diet supplemented by 20,000 or more units of vitamin A never produced a startling cure. The most rapid recovery of a normal threshold occurred in 2 subjects and required four weeks of an adequate diet. In most cases, however, the return of the visual threshold to normal took much longer and in some instances was not accomplished in three months.

Vitamin A is stored in the liver, and disturbances of the function of this organ may be expected to influence the visual threshold. Measurements of the dark adaptation of several patients with alcoholic cirrhosis of the liver showed changes resembling those of acute dietary avitaminosis A. These symptoms were removed by supplementing the patients' diets with large amounts of vitamin A.

The conclusion of all this is that measurements of dark adaptation when made under standard and precise conditions constitute a tool for discovering dietary deficiencies and disease conditions concerned with a deficiency of vitamin A.

The researches on which this lecture was based were made in collaboration with Drs. Joseph Mandelbaum, Charles Haig, Arthur J. Patek Jr. and Simon Shlaer. For specific references to the points briefly abstracted here, the reader is referred to a recent paper entitled "Relation Between Vitamin A and Dark Adaptation" (*J. A. M. A.* 112: 1910-1916 [May 13] 1939).

Clinical Aspects of Vitamin A and Dark Adaptation. DR. FRANCIS HEED ADLER, Philadelphia.

DISCUSSION ON PRESENTATIONS BY DRS. HECHT AND ADLER

DR. ROBB McDONALD, Philadelphia: I feel that it is rather presumptuous of me to say anything after hearing these two presentations; my only excuse for doing so is that I have been using Dr. Hecht's apparatus for over a year and have been interested in studying the physiologic basis of dark adaptation. However, I should like to show two slides and to discuss some work I am doing with Dr. Adler in the department of ophthalmology at the University of Pennsylvania and how we hope to get to the basis of this problem.

The first slide depicts the electrical response in a single fiber of the optic nerve of a horseshoe crab, which goes to show that we are getting down to the basis of the visual response. This slide has nothing to do with dark adaptation but shows the effect of intensity of stimulation on the discharge. The spikes are action potentials, and the response varies with the intensity of illumination. The brighter the light, the shorter the latency and the greater the frequency of the response.

The next slide shows the change in the response to dark adaptation. Here again are demonstrated action potentials. There is a constant intensity of stimulus and a constant time of exposure. In the early responses after light adaptation there is a latency which is definitely longer than the latency shown after an hour, and the number and the frequency of impulses increase with dark adaptation. We have accumulated considerable data on this objective method, and I think that shortly we will be able to show that the character of the response in the dark-adapted state is not the same as in the light-adapted state. We also hope to clear up some other problems in the physiology of dark adaptation.

DR. BENJAMIN FRIEDMAN: I should like to ask Dr. Hecht, first, whether during the course of the induced vitamin A deficiency his patients were able to distinguish the effects of this deficiency clinically, and, second, whether he has found any evidence to support the contention

that the brain is also a factor in dark adaptation or whether he feels that this is solely a peripheral phenomenon.

DR. SELIG HECHT: In answer to Dr. Friedman's first question, our subjects reported almost no subjective findings in their daily life. They were questioned regularly, and their fields were watched and their eyes examined; however, with the exception of 1 patient they reported nothing. This patient showed the most extreme response to the vitamin A deficiency. His threshold had increased nearly 2 logarithmic units. One day he said, "Last night I bumped into a wall which I should have seen but did not." That was the first report made of any subjective recognition that all was not normal. Since the threshold was 2 logarithmic units above normal, we became apprehensive and terminated the experiment, putting the subject on a normal diet, supplemented with vitamin A.

There has been a good deal of loose talk about vitamin A and night driving, and I may say from my own experience that it is largely unfounded. One of the subjects of these experiments, Dr. Mandelbaum, did a great deal of night driving and found no difficulty with it, undoubtedly because under ordinary circumstances the illumination that prevails is never near the threshold. Please remember what near the threshold means. It represents an extremely small amount of light, and I suppose if one were expected to function at this very margin of illumination, one would be sure to be affected and to notice an incapacity to see. But in the city and even in the country one is not subjected to that small amount of light.

Perhaps I may be pardoned if I tell the details of an experience with a colleague who was sensitive about his vision. He stated that he was certain he could tell merely by his night driving experience when he was getting an adequate amount of vitamin A and when he was not. His dark adaptation was measured when, according to his judgment, he was in fine condition, and a normal curve was obtained. I told him that the first time he felt he was in poor condition to come in for another test. He called up one day and said that he was in miserable shape. His dark adaptation was found to be precisely what it was before. The effect of vitamin A on night driving was reported by Jeghers in Boston. However, he used the biophotometer as the method of measuring the threshold, and this instrument used as he did is rather unreliable, to put it mildly. One cannot tell what it shows, because it is hard to say just what it measures.

The other point to which Dr. Friedman refers is probably the work of Elsberg, who insists that dark adaptation takes place in the brain. I am sure that Dr. McDonald's measurements which have just been shown demonstrate that it does not take place in the brain. These retinal potentials were taken from the isolated eye, freed from the brain; nevertheless they show adaptation of the same kind and order of magnitude as in the intact animal. Moreover, protozoa, clams, ascidians and even plants adapt themselves to darkness.

DR. WENDELL L. HUGHES: Have any measurements been made on the other eye when one eye is dark adapted? If so, I should like to know whether dark adaptation in one eye made any difference in the opposite eye. This remark is made with reference to Dr. Elsberg's work, reported here last year.

DR. SELIG HECHT: There has been a great deal of discussion as to whether the threshold with two eyes is the same as with one eye, and the problem is not settled. In this work the eyes can be measured alternately or simultaneously, and the result is practically the same. I could be more specific, since I know what Dr. Hughes is asking for, but I think I will let it go at that.

DR. E. M. JOSEPHSON: I should like to ask Dr. Hecht if the superfine dark adaptation test which he has used in his work is at all applicable clinically. Dr. Adler has shown that the dark adaptation may vary considerably in various areas in the retina. Clinically I have the feeling that it would be almost impossible to have a patient hold his eye so quiet for thirty minutes of testing or for one to get absolutely identical spots for repeated successive examinations. I have tried to do so, without much success. (The biophotometer, I have found, gives the clinically valuable data on the dark adaptation of the entire eye, in spite of its failure to yield ultrascientific data on the reactions of a single rod or cone.)

DR. FRANCIS HEED ADLER, Philadelphia: It is true that fixing is the *bête noire* of the whole problem, and I can say with confidence that the reason Dr. Hecht is so vehement about the biophotometer is that fixation is extremely poor with that instrument. With Dr. Hecht's instrument it is true the subject has to cooperate, but one does have more control of the fixation with that instrument than with any other I know of, and I agree that one can tell fairly accurately just what portion of the retina is being investigated.

DR. JOSEPH MANDELBAUM: The subject is not required to look at the fixing point and hold the fixation for thirty minutes. He is required to do so only during the minute or two that it takes to make a reading. In the interval, the subject can sit back and relax.

There was a point which Dr. Adler mentioned about corneal dystrophy. I have examined 3 patients with this condition. Instead of using the affected eye to record dark adaptation, I used the normal eye. Although there were no opacities which might account for it, I obtained deficient dark adaptation in 2 of these cases. I do not know whether vitamin A deficiency causes corneal dystrophy or not.

DR. JOSEPH IGRSHEIMER, Istanbul, Turkey: Behr has pointed out that the question of dark adaptation is important in the differential diagnosis between choked disk and optic neuritis. It would interest me to know whether Dr. Adler has had the same experience.

DR. FRANCIS HEED ADLER, Philadelphia: I have not, but I should take it for granted that the dark adaptation curve has the same value as the visual acuity test, which is one of the chief means of differentiating between neuritis and papilledema.

DR. FRANK D. CARROLL: With reference to Dr. Igersheimer's question, if a scotoma is present in the area tested, the dark adaptation curve is elevated. I have tested numerous persons with toxic amblyopia with the adaptometer devised by Dr. Hecht and have found in the area corresponding to the scotoma a definite elevation of the curve, whereas if another place on the retina is tested it may show no abnormality. As the toxic amblyopia gets better, the scotoma disappears and the curve for the area returns to normal.

DAVID H. WEBSTER, M.D., *Chairman*

ROBERT K. LAMBERT, M.D., *Secretary*

Jan. 15, 1940

Motion Picture Demonstration of Unusual Neuro-Ophthalmologic Conditions. DR. S. PHILIP GOODHART and DR. BENJAMIN BALSER.

DISCUSSION

DR. S. P. GOODHART: As Dr. Balser has stated, we are preparing an atlas for cinematographic demonstration of various forms of diseases of the nervous system. This atlas, describing the individual films for the instruction of students, will be made available to medical schools. The film presented here is one of this collection. In this film, as in our others, we present rather rare forms of ocular conditions with associated pathologic conditions of the central nervous system. All ophthalmologists are familiar with the intimate relation between the mechanism governing the function of the ocular structures and that governing the function of the central and peripheral nervous systems. Among the clinical syndromes presented here, that called "skew deviation" is one resulting from pathologic involvement of the brain stem; in the case presented it is of interest to note that a neoplasm of the posterior fossa caused pressure anteriorly and that the resulting ocular condition was relieved on removal of the neoplasm.

The study of three different forms of nystagmus is of clinical interest, as each form is shown as a part of a syndrome of extensive organic pathologic involvement and yet the result of a minute lesion, involving in each instance the posterior longitudinal fasciculus. The cases presented are of clinical as well as of anatomic interest and indicate the importance of evaluating a discrete pathologic process which makes its presence known by an easily overlooked clinical sign. Nystagmus, especially in cases of multiple sclerosis, usually affects both eyes.

The case of myasthenia gravis illustrated here is another example of the intimate association between ocular manifestations and more extensive disease within the nervous system. The patient came to the hospital complaining only of general weakness, and examination brought out occasional difficulty in fully raising the lids. Transitory muscular weakness and fatigue so characteristic of the disease were evident when the patient was asked to follow an object—the examiner's finger—as it moved rapidly in extreme lateral positions; one then observed a slowly increasing degree of ptosis with final closure of the eye due to the failing power of the levator muscles of the upper lids. With a few minutes of rest, power was recovered. The point is that only careful testing of the function of the ocular muscles gave the early objective findings of a serious neurologic condition.

The last condition demonstrated in the film, neuromyelitis optica, is a rare clinical entity. The clinical picture may show as its early manifestation beginning atrophy of the optic nerves, while the objective findings referable to the involvement of the spinal cord may be overlooked. This case again illustrates the close relation of the two branches

of medicine, ophthalmology and neurology, as demonstrated by some of the more unusual forms of neuropathologic involvement.

An Operation For Spastic Entropion. DR. RAYMOND E. MEEK.

This paper will be published in full, with discussion, in a later issue of the ARCHIVES.

Central Scotomas: Their Importance in Topical Diagnosis. DR. FRANK B. WALSH and DR. FRANK R. FORD.

This paper will be published in full, with discussion, in a later issue of the ARCHIVES.

A New Corneoscleral Suture. DR. JOHN M. McLEAN.

This paper was published in full in the March issue of the ARCHIVES, page 554.

DISCUSSION

DR. RAMON CASTROVIEJO: I should like to ask Dr. McLean three questions: In the series of 110 cases in which his sclerocorneal suture was used were there any instances in which either the suture or its scleral attachment was cut while making the incision? How many days after the operation is the patient allowed to get out of bed? How many days after the operation is the eye which is not operated on uncovered?

DR. BENJAMIN FRIEDMAN: In tying the suture I noticed that there was a tendency for the lower lip of the wound to become everted. This is due to the difficulty in pulling the suture through the relatively thick cornea. I should like to ask whether reversing the direction of the suture would not tend to obviate this difficulty.

DR. JOHN M. McLEAN: Dr. Castroviejo asked if I ever cut the suture in making the section. Yes, I did that once. I do not think there is any excuse for it, but I did it. However, if I do cut the suture, I still have a prepared conjunctival flap, so if I care to I can use ordinary conjunctival sutures or, as I did in the case in which I cut the suture, I can rethread those tracks after Verhoeff's technic.

In regard to how many days after the operation I let the patient get up, that is variable. In that connection I might relate something about the first motion picture I showed. From the technical standpoint, it is not a good film. The patient was not cooperative. She was rolling her head all over the table, and I was following her, and the motion picture camera was following us both. She was a charming old lady from the Old People's Home and very, very senile. The night after her operation she became thoroughly confused and stood up in bed. Before the nurse in the ward could reach her she executed a practically perfect swan dive from the bed to the floor, striking the eye that had been operated on on the corner of the table. The intern had to use a half a dozen sutures to repair the laceration of her upper lid, but she did not burst the wound open, and her eye healed uneventfully. This incident represents one extreme in getting the patient up. I have had some old mountaineers from West Virginia who will not get up, but as a general rule I try to get the patients up as early as possible. I have never had any trouble attributable to getting them up too early when this

suture was used. In that respect I am held back a little because the general hospital routine is that which has gone on for many years. I should like to get these patients up as early as Dr. O'Brien does, the day after they are operated on or shortly thereafter, but that I have not been able to do routinely. The third question concerns the eye that is not operated on; the standard technic at the Wilmer Ophthalmological Institute, which has held over from the days when purely conjunctival flap sutures were used, is to leave both eyes bandaged for four days, and to unbandage the eye that is not operated on on the fourth day; however, if the patient becomes confused I unbandage this eye immediately in order to keep the patient in touch with reality as far as possible.

Dr. Friedman wants to know about reversing the suture and putting it in the other way around. I do not think it makes much difference. The needle is going to drag a little when it goes through the cornea. It drags a little when the Liégard or Verhoeff or any other technic is employed. I put the suture in this way because it was a little easier to see what I was doing. Is that what Dr. Friedman means?

DR. B. FRIEDMAN: No, I refer to the final stage when the suture is tied. One has to pull the suture through the corneal wound in a downward direction, which at the time the vitreous is exposed causes an undesirable gaping.

DR. JOHN M. McLEAN: I do not do that ordinarily. When the operation is being photographed, in order to keep my hand from obscuring the view the hand is backward, and exactly the same thing is done in tying the suture at the end. I do tie it the way Dr. Friedman suggests unless I am trying to illustrate the procedure by motion pictures, and then I have to get my hand out of the way as far as possible. I have never had any trouble through loss of vitreous while tying sutures.

DR. JAMES W. SMITH: To what depth is the corneal slot fashioned with the Lundgard knife and what is the width of the suture material used in the flap?

DR. JOHN M. McLEAN: The slot goes approximately halfway to the anterior chamber. For practical purposes it goes deep enough to get the needle through. The needle is known as Kalt's corneal needle. I cannot say just what is the size of the suture material, because it is sold under the name of Kalt's corneal silk, and I do not think the size is given.

COLLEGE OF PHYSICIANS OF PHILADELPHIA,
SECTION ON OPHTHALMOLOGY

ALEXANDER G. FEWELL, M.D., *Chairman*

WARREN S. REESE, M.D., *Clerk*

Dec. 21, 1939

Transcranial Extirpation of a Fibrohemangioma of the Orbit:
Report of a Case. DR. BENJAMIN SOUDERS.

This article will appear in full in a later issue of the ARCHIVES.

The Extraocular Muscles in Myasthenia Gravis and Hyperthyroidism.

DR. MELVIN W. THORNER.

Palsy of the extraocular muscles is so commonly present in cases of myasthenia gravis that many patients with this condition are seen by the ophthalmologist first. The paralysis is characterized chiefly by the facts that it frequently changes from one muscle to another and that the weakness is greatly lessened by the administration of prostigmine. The current most generally accepted explanation of myasthenic weakness is that there is an area of deficient conduction of the nerve impulse at the myoneural junction. This (according to Dale) is partly dependent on the presence of acetylcholine at this junction. According to this hypothesis, acetylcholine is either destroyed too rapidly or is produced in insufficient quantity in the myasthenic patient. More recent work would indicate that the myoneural junction is not the sole site of the pathophysiologic disturbance in myasthenia gravis. The pathologic changes are not prominent. There are intramuscular collections of lymphocytes (the lymphorrhages), which are found most frequently in the extraocular muscles. Also, it should be noted that thymic abnormalities, chiefly neoplasms, are found at 50 per cent of autopsies.

In the case of hyperthyroidism a totally different situation exists. Approximately 50 per cent of patients with hyperthyroidism may present some degree of exophthalmos, although it may be necessary to use an ophthalmometer to detect the milder degrees. While the ultimate cause of exophthalmos is unknown, the proximate cause is hypertrophy of the orbital contents. In many cases this hypertrophy and mechanical distortion are so great that ocular movements are impeded. The explanations for exophthalmos that have been advanced are: 1. It is caused by excessive thyroid secretion. 2. It is caused by sympathetic stimulation of Müller's muscle. However, experimental evidence has shown that exophthalmos may be produced in thyroidectomized and sympathectomized animals by the administration of an anterior pituitary extract containing the thyrotropic hormone. The existence of true isolated extraocular palsies in hyperthyroidism is rare, occurring in 1 of 300 cases. The pathologic picture of hyperthyroidism consists of fairly constant lymphorrhages in the extraocular muscles and hypertrophy of the thymus gland; the latter change is not usually associated with the formation of a neoplasm.

The concomitant association of these two conditions in the same patient is an interesting phenomenon. When it occurs, as it rarely does, a type of "see-saw" balance is established between the two. When the hyperthyroidism is most marked, the myasthenic symptoms are least marked. It was possible in such a case (Relation of Myasthenia Gravis to Hyperthyroidism, *Arch. Int. Med.* 64: 330 [Aug.] 1939) to establish a therapeutic balance for a time.

This presentation has been an attempt to show the difference in mechanism by which ocular movement is impaired in cases of myasthenia gravis and of hyperthyroidism. In addition, the mutual antagonism of the conditions, when they coexist, has been noted.

DISCUSSION

DR. I. S. TASSMAN: I should like to ask Dr. Thorner his explanation for the various muscles being affected at different times and for one

muscle recovering when another becomes affected, only to become again involved at a later stage.

It might also be of interest to know whether the effect of prostigmine in myasthenia gravis is produced by its action on the choline esterase, whereby it would serve to interfere with the esterase in its role as antagonist to the parasympathetic activity of acetylcholine.

DR. WARREN S. REESE: I should like to ask Dr. Thorner whether it is true that in these cases of myasthenia gravis the paralysis increases toward the end of the day.

DR. H. MAXWELL LANGDON: There is no doubt that patients with myasthenia gravis show more marked changes toward the end of the day than they do in the early morning. I have seen about 4 patients with this condition, and all of them showed less activity toward the end of the day not only in the ocular muscles but in other parts of the body.

The word "paralysis" in relation to these cases does not seem very accurate to me, although I suppose it is the best word available to describe the condition of the patients.

Myasthenia, suggesting a fatigue of muscles, is descriptive, and the condition does progress to actual loss of muscular function. Therefore, it seems to me that paralysis is not inexact but is rather inaccurate.

I was interested in the rather lengthy comparison of myasthenia gravis with hyperthyroidism. To me, the conditions are so different that if one has seen only a case or two of each there should be no difficulty in making a differential diagnosis.

DR. MELVIN W. THORNER: Dr. Tassman's question, as he probably suspected, is not easy to answer. In myasthenia, each muscle appears to have a certain limited rate of energy production. When this is used up, as for example in an external rectus muscle, it may no longer function until it has had a recovery period. During this recovery period, the internal rectus muscle may become exhausted and inactive. In this manner a plausible explanation for the rotation of weakness of the extraocular muscles may be built up.

While prostigmine is postulated either to inhibit the abnormally great destruction of acetylcholine (by cholinesterases) or to stimulate its production, neither of these two hypotheses is definitely proved. The blood esterase findings have not as yet shown sufficient agreement among investigators to afford a basis for a pedantic and categorical statement.

The aforementioned facts concerning the limited energy production in muscles might serve to explain why the myasthenic patient is weaker at night. The day's endeavors would be likely to have used up most of the available energy supply, so that at night the weakness is most marked. This clinical fact was noted by Erb and Goldflam and most of the clinicians following them, but this rule, as most others, has its exceptions. Some patients are worse in the morning than at night.

I agree with Dr. Langdon's abhorrence of the word "paralysis." Most generally, the term would broadly include any one of many conditions which have in common the inability to perform a movement voluntarily. The unsatisfactory word in such a definition is the word "voluntary," which could lead to a host of esoteric and philosophic arguments. All this is, of course, pointless when the mechanism underlying a "paralysis" is known.

A Clinical Evaluation of Dark Adaptation Tests. DR. FRANCIS H. ADLER and DR. ROBB McDONALD.

This paper will appear in full, with discussion, in a later issue of the ARCHIVES.

A Clinician's Experience with Sulfanilamide. DR. BURTON CHANCE.

The results of sulfanilamide therapy in a group of cases, including cases of trachoma, uveitis and exfoliative keratitis, is reported. Only small doses were taken by mouth, with remarkable effects. The almost miraculous alleviation of all symptoms which resulted should be ascribed solely to sulfanilamide.

Book Reviews

Hydrophthalmia or Congenital Glaucoma. Its Causes, Treatment and Outlook. By J. Ringland Anderson, M.C., M.D., B.S. (Melb.) F.R.C.S. (Edin.), F.R.A.C.S., D.O.M.S. (Lond.), Ophthalmic Surgeon to Alfred Hospital, Melbourne. With a foreword by Sir John Herbert Parsons, C.B.E., D.Sc., F.R.C.S., F.R.S. Price, 25 shillings net. Pp. 377, with 116 illustrations. Published for the British Journal of Ophthalmology, by the Cambridge University Press, London, 1939.

This monograph is one of the publications sponsored by the *British Journal of Ophthalmology*. It is a handsome volume, profusely and beautifully illustrated.

The subject of hydrophthalmia is exhaustively treated under the chapter headings of general etiology, differential diagnosis, the structure and development of the involved tissue, their embryology and comparative anatomy, the pathology of congenital glaucoma, pathogenesis, treatment, prognosis and general reflections.

The terms congenital glaucoma and hydrophthalmia are preferred by the author. He suggests that the use of all other terms be abandoned. The author states that "the condition to be described is that of an eye which has become enlarged under the influence of increased tension." Subsequently, he says that "the ocular condition that we are to consider is that which is due to a rise in tension occurring at so early an age that the coats of the globe distend." The latter definition seems to be more appropriate.

He considers that no hard and fast line divides the congenital, juvenile and adult forms of glaucoma. In the chapter on differential diagnosis, the condition of megalocornea is fully described.

In the study of the comparative anatomy of the parts involved, the author states that in the human eye the ciliocleral sinus is generally so empty of uveal tissue that it appears like part of the angle. The disappearance of this meshwork gives the ciliary muscle maximum freedom but may be a real loss to the eye, if the supposition is right that a true pectinate ligament aids the circulation of aqueous by capillary attraction and by preventing the iris root from coming in contact with the cornea.

In 86 per cent of twenty-eight specimens of early hydrophthalmia, the cause of the condition was probably the fetal state or the abnormal development of the meshwork of the angle.

As to treatment of the local condition, in certain cases miotics appear to be beneficial and are therefore worthy of trial, but the author adds "the result is determined by the actual pathological state."

The author states that regarding operation one may say that any hope for success almost vanishes after the first year. The superiority of Elliot's operation is possibly masked by the fact that it is more or less in general use and therefore performed by more inexpert operators. Much can be hoped from the operation described by Barkan, for this

gives the surgeon a reasonable chance of establishing a channel of escape through the obstructing tissue at the angle by making an incision under direct and magnified vision.

The pocket attached to the inside of the backboard contains tables of pathologic observations in the author's cases and in those reported in the literature. A commendable feature is the summary or conclusions and the references at the end of each chapter. The subject matter is brought up to date. While the book is primarily a reference book, the chapter on treatment contains a critical consideration of all the different procedures, with an analysis of results.

The book will prove a valuable addition to the library of all students of ophthalmology.

WILLIAM ZENTMAYER.

Traité d'ophtalmologie. Published under the auspices of the Société française d'ophtalmologie, by P. Bailliant, C. Coutela, E. Redslob, E. Velter and R. Onfray, general secretary. Eight volumes, pp. 8,058, with 176 plates in color. Subscription unbound edition, 2,500 francs; bound edition, 2,800 francs. Single volume, 350 or 400 francs. Paris: Masson & Cie, 1939.

Volumes I and II were reviewed in the October 1939 issue of the ARCHIVES, page 713.

Volume III (1,146 pages, 460 figures, 9 plates in color) contains three subdivisions with the following titles: "Technic" (continued), "Visual Anomalies" and "Pathology."

In the part on technic of the laboratory the following subjects are considered: chemical and serologic examination of the blood, spinal fluid and intraocular fluid, by A. Dubois-Poulsen; bacteriology by L. Carrère; elements of pathologic technic by L. Carrère, and animal parasites of the eye, by G. Senevet.

Motility of the eyeball and binocular vision are then described under the following headings: "Examination of Motility of the Eyeball," by Jean Nordmann, and "Examination of Binocular Vision," by René Onfray.

The second main subdivision, "Visual Anomalies" is divided into chapters with the following titles: "Hypermetropia," "Astigmatism" and "Anisometropia," by H. Joseph; "Myopia," by Jacques Mawas; "Presbyopia and Troubles of Accommodation," by L. Hambresin; "Vision and Correction of Aphakic Eyes," by Emile Haas; "Anomalies of Light Sense," by F. Bourdier, and "Anomalies of Color Sense," by A. Polack.

The principal subdivision in this volume is designated "Pathology" and is continued in volumes IV, V and VI. The subject is introduced by a consideration of general pathologic and biologic reactions with special application to the eye, which are considered under the following headings: "Pathology of the Cell" and "Inflammation" and "Tumors," by E. Redslob and L. Gery. This chapter of 174 pages is a most instructive presentation based on the point of view of general pathology. Chapters entitled "Ocular Allergy," by André Koutseff, and "Vitamins," by Nicholas Bezssonoff, are of great interest at the present day. Professor Terrien writes on the relation of general pathology to ophthalmology. A section entitled "Glands of Internal Secretion in Their Relation to

Ophthalmology" (100 pages) is from the pens of A. Franceschetti and G. Gorin. General ophthalmologic symptoms are reviewed by F. Terrien in a section which forms an introduction to the parts that follow, in which the diseases of each anatomic part are taken up in turn: diseases of the eyebrow, by H. Villard; diseases of the lids, by E. Aubaret; tumors of the lids, by Marcel Kalt; tumors of the caruncle, by Marcel Kalt, and disturbances of the motor apparatus of the eyelids, by Jean Sédan.

In volume IV (898 pages, 233 figures and 36 plates in colors) the general heading "Pathology" is continued, and the following conditions are taken up: lacrimal apparatus, by V. Valière-Vialeix; diseases of the conjunctiva, by G. Renard and Roger Nataf; trachoma, by A. Cuénod and Roger Nataf; conjunctival lesions occurring in general diseases or in diseases of the neighboring organs, lesions of the conjunctiva produced by certain chemical substances or by substances of animal or vegetable origin and conjunctival lesions produced by physical agents—light, radium, x-ray and electricity, by L. Genet; tumors of the conjunctiva, by Mériqot de Treigny; diseases of the cornea, by P. Prélat; other diseases of the cornea, by G. Kleefeld, and diseases of the sclerotic, by Paul-J. Petit.

The chapters on conjunctival diseases (G. Renard and Roger Nataf) and on trachoma (A. Cuénod and Roger Nataf) are outstanding, not only for the excellence of the scientific approach in the text but for the illustrations, many of which are in color.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

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OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
 Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.
 Place: Perth, Western Australia. Time: Sept. 2 and 7, 1940.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
 Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
 All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
 Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
 Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
 Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England
 Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
 Time: July 4-6, 1940.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arie Feigenbaum, Abyssinian St. 15, Jerusalem.
 Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
 Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
 Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
 Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIEDADE DE OPHTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Francisco Ferreira, Pitangueiras 15, Brotas, S. Salvador, Brazil.
 Secretary: Dr. Adroaldo de Alencar, Brazil.
 All correspondence should be addressed to the President.

SOCIETÀ OTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
 Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo. 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7°.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
 Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arich-Friedman, 96 Allenby St., Tel Aviv, Palestine.
 Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON
OPHTHALMOLOGY

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
 Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
 Place: New York. Time: June 10-14, 1940.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY,
SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
 Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts
 Bldg., Omaha.
 Place: Cleveland. Time: Oct. 6-11, 1940.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
 Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
 Place: Hot Springs, Va.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
 Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
 Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.
 Place: Royal York Hotel, Toronto. Time: June 19-21, 1940.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
 Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.
 Executive Director: Mrs. Eleanor Brown Merrill, 50 W. 50th St., New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON
EYE, EAR, NOSE AND THROAT

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
 Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
 Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of
 each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Nadeau, 122 E. Walnut St., Green Bay.
 Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
 Place: The Gateway Inn, Land O'Lakes. Time: June 1940.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
 Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
 Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time:
 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick G. Sprowl, 421 Riverside Ave., Spokane, Wash.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: Spokane, Wash. Time: June 24-27, 1940.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Clarence W. Shannon, 4th and Pike Bldg., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1215-14th Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. A. Shultz, 303 N. Main St., Rockford, Ill.
Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIoux VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Dake Biddle, 123 S. Stone Ave., Tucson, Ariz.
Secretary: Dr. M. P. Spearman, 1001 First National Bank Bldg., El Paso, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Virgil Payne, Pine Bluff.
Secretary-Treasurer: Dr. Raymond C. Cook, 1005 Donaghey Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Melville Black, 424 Metropolitan Bldg., Denver.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR,
NOSE AND THROAT

President: Dr. Shirley H. Baron, 309 State St., New London.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Grady E. Clay, 384 Peachtree St. N. E., Atlanta.

Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.

Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.

Place: Indianapolis. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.

Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Place: Davenport.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. D. C. Montgomery, 301 Washington Ave., Greenville, Miss.

Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Place: St. Charles Hotel, New Orleans. Time: April 25, 1940.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY

Chairman: Dr. O. B. McGillicuddy, 124 W. Allegan St., Lansing.

Secretary: Dr. A. R. McKinney, 330 S. Washington Ave., Saginaw.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Hendrie W. Grant, 330 Lowry Medical Arts Bldg., St. Paul.

Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.

Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Roy Grigg, Bozeman.

Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY,
OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. James S. Shipman, 542 Cooper St., Camden.

Secretary: Dr. Wright McMillan, 23 Passaic Ave., Passaic.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.

Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. G. Woodward, 100 S. James St., Goldsboro.

Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.

Secretary-Treasurer: Dr. F. L. Wicks, 516-6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.

Secretary-Treasurer: Dr. R. S. Fixott, 1020 S. W. Taylor St., Portland.

Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
 Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
 Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m.,
 second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
 Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
 Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg.,
 Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. J. W. Ward, 2607½ Lee St., Greenville.
 Secretary: Dr. Dan Brannin, Medical Arts Bldg., Dallas.
 Place: Fort Worth. Time: December 1940.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. A. E. Callaghan, Boston Bldg., Salt Lake City.
 Secretary-Treasurer: Dr. Rowland H. Merrill, 1010 First National Bank Bldg.,
 Salt Lake City.
 Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of
 each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
 Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd. S. W., Roanoke.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE
 AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
 Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
 Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
 Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Arthur G. Fort, 478 Peachtree St. N. E., Atlanta, Ga.
 Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
 Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month
 from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital,
 Baltimore.
 Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
 Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m.,
 fourth Thursday of each month from October to May.

BIRMINGHAM EYE, EAR, NOSE AND THROAT CLUB

President: Each member, in alphabetical order.

Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.

Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.

Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.

Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.

Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.

Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.

Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.

Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.

Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.

Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI OPHTHALMIC CLUB

Chairman: Each member, in rotation.

Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.

Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

CLEVELAND OPHTHALMOLOGIC CLUB

Chairman: Dr. Albert J. Ruedemann, Cleveland Clinic, Cleveland.

Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.

Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.

Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.

Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Harry M. Sage, 9 Buttles Ave., Columbus, Ohio.

Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.

Place: The Neil House. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.

Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.

Time: Second Friday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Dan Brannin, Medical Arts Bldg., Dallas, Texas.
 Secretary: Dr. L. E. Darrough, 4105 Live Oak St., Dallas, Tex.
 Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
 Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
 Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
 Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
 Time: 6:30 p. m., first Wednesday of each month.

DETROIT OPHTHALMOLOGICAL SOCIETY

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
 Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
 Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
 Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
 Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
 Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
 Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
 Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
 Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. J. Charles Dickson, 1617 Medical Arts Bldg., Houston, Texas.
 Secretary: Dr. William J. Snow, 708 Medical Arts Bldg., Houston, Texas.
 Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
 Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
 Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.

Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.

Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.

Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.

Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.

Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE AND EAR SOCIETY

President: Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.

Secretary-Treasurer: Dr. J. W. Fish, Brown Bldg., Louisville, Ky.

Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St. N. W., Washington.

Secretary: Dr. Elmer Shepherd, 1606-20th St. N. W., Washington.

Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.

Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Herbert G. Smith, 411 E. Mason St., Milwaukee.

Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.

Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.

Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.

Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.

Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St. W., Montreal, Canada

Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, 119-7th Ave., Nashville, Tenn.

Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.

Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW HAVEN OPHTHALMOLOGICAL SOCIETY

President: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. David H. Webster, 140 E. 54th St., New York.
Secretary: Dr. Robert K. Lambert, 10 101-15th Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Arthur M. Yudkin, 257 Church St., New Haven, Conn.
Secretary: Dr. Benjamin Esterman, 515 Park Ave., New York.
Place: Squibb Hall, 745-5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND
OTO-LARYNGOLOGICAL SOCIETY

President: Dr. J. T. Maxwell, 1140 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner;
7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President. Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Adolph Krebs, 509 Liberty Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351-5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. J. F. Hardesty, Missouri Theatre Bldg., St. Louis.

Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.

Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.

Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.

Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE,
EAR, NOSE AND THROAT

Chairman: Dr. Matthew Hosmer, 384 Post St., San Francisco.

Secretary: Dr. Fred Boyle, 490 Post St., San Francisco.

Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. I. Henry Smith, Slattery Bldg., Shreveport, La.

Secretary-Treasurer: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.

Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter W. Henderson, 407 Riverside Ave., Spokane, Wash.

Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.

Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.

Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.

Place: University Club. Time: First Tuesday of each month except June, July and August.

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. S. H. Patterson, 1251-15th St., Toledo, Ohio.

Secretary: Dr. J. E. Minns, 316 Michigan St., Toledo, Ohio.

Place: Toledo Club. Time: Each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.

Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.

Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. Ernest Sheppard, 927 Farragut Sq. N. W., Washington, D. C.

Secretary-Treasurer: Dr. E. Leonard Goodman, 1801 I St. N. W., Washington, D. C.

Place: Episcopal Eye and Ear Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

Chairman: Each member in turn.

Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.

Place: Office of chairman. Time: Last Tuesday of each month from October to May.

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Continuing the Publication

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Knapp's "Intraocular Tumors," with the author present. At the head of the part devoted to the scientific proceedings of 1871 there is the impressive inscription, outlined in black.

*In Memory of
Albrecht von Graefe
whose life and works
so greatly helped to
turn a doubtful specialty
into a certain science.*

When the monument was erected in Berlin to von Graefe, members of the society sent a contribution through H. W. Williams, which was acknowledged by the treasurer, Felix Mendelssohn.¹⁶

The society has made steady progress. Its membership, which was 48 in 1870, has risen to 180, as is shown in the accompanying chart.¹⁷ But the total number elected in these seventy-five years has been 448. Of this number 268 have died or resigned.

During these seventy-five years meetings have been held annually except in 1867 (Niagara Falls), when there was no quorum, and in 1872, when the meeting was called off because of the International Ophthalmological Congress in London. But in 1888 a second fall meeting was held in Washington, D. C., during the first Congress of American Physicians and Surgeons. The average attendance at the meetings is about 44 per cent during the entire period. The place of meeting varied as follows: New York (1864, 1865, 1876 and 1906), Boston (1866 and 1905), Niagara Falls (1867 and 1877), Newport, R. I. (1868, 1869, 1870, 1871, 1873, 1874, 1875, 1878, 1879, 1880 and 1881), Lake George, N. Y. (1882), Catskill, N. Y. (1883, 1884 and 1890), New London, Conn. (1885, 1886, 1887, 1888, 1889, 1892, 1893, 1895, 1896, 1898, 1899, 1901, 1902, 1908, 1909, 1911, 1915, 1918 and 1932), Washington, D. C. (1891, 1894, 1897, 1900, 1903, 1907, 1910, 1913, 1916, 1922, 1925, 1928 and 1933), Atlantic City, N. J. (1904, 1912 and 1919), Hot Springs, Ark. (1914, 1917, 1920, 1924, 1926, 1929, 1930, 1935, 1936 and 1937), Swampscott, Mass. (1921), Colorado Springs, Colo. (1923), Quebec, Canada (1927), Asheville, N. C. (1931), Lucerne, Quebec, Canada (1934), and San Francisco (1938).

16. Minutes of the Proceedings of the Annual Meeting, Tr. Am. Ophth. Soc. 2:122, 1874.

17. The membership, long limited to 200, was raised to the limit of 225 in 1926.